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OBSERVATIONS ON THE USE OF FLUORINE FOR THE PARTIAL CONTROL OF DENTAL CARIES.[1]

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METHODS OF APPLICATION OF FLUORINE.

THE commonest methods of application of fluorine are (i) fluoridation of domestic water supplies and (ii) topical application of the fluoride.

1. Fluoridation of Domestic Water Supplies.

Public health has progressed at an impressively rapid rate in the last two hundred years. Some of the major advances in practice are referable to Jenner's anti-smallpox vaccine, to Pasteur's advancement of the microbial theory of disease, to water and sewerage sanitation for the control of intestinal infections, to pasteurization of milk for the elimination of milk-borne diseases, to antimosquito measures for the control of malaria, to immunization against diphtheria and to the administration of iodine for goitre prevention. What is claimed (Bull, 1952) to be the most important discovery in public health practice in the last twenty-five years, and one ranking with some of those specified above, is the fluoridation of domestic water supplies for the partial control of dental decay.

Historical Survey.

In 1901 Eager observed in Italian emigrants a condition of the teeth known as "*denti di Chiaie*"; he attributed it to local geological conditions and noted that a change of water supply lowered the incidence of the condition.

In 1916 Black and McKay observed the incidence of mottled enamel in certain areas. They incriminated some unknown substance present in the drinking water and used during the period of calcification of the teeth.

In 1931 Churchill and Smith, Lanz and Smith (in the United States), and Velu and Balozet (in North Africa) independently proved that waters which caused these abnormalities in the teeth contained fluorine.

Dean *et alii* in a series of papers (1938) showed conclusively that the presence in domestic water supplies (through natural agencies) of one to two parts per million of fluorine was associated with a lowered incidence of dental caries, and that this improvement in dental health was of considerable magnitude.

To test the correctness of the deductions made from the observations outlined above and to ascertain the possibility of their practical application, the cities of Newburgh and Kingston, New York, United States of America, agreed to institute a scheme of artificial fluoridation of the city water

[1] This paper is based on an address delivered to the twenty-ninth meeting of the Australian and New Zealand Association for the Advancement of Science (Section I) held in Sydney in August, 1952.

supply of Newburgh. Kingston, a community similar in almost every respect to Newburgh, was to act as control by continuing the use of its fluoride-free water. The plan was put into effect in May, 1945, and has been consistently followed ever since. Another experiment had been set up at Grand Rapids and Muskegon (control), Michigan, a few months earlier (January, 1945) (Faber, 1951).

Before any such practice can be enforced or recommended or even accepted, it must be proved to be (i) harmless, (ii) efficacious, and (iii) practicable. Great care was devoted to devising the details of the Newburgh-Kingston experiment, not only to prove the benefit derived from the use of fluoridated water, but also to demonstrate the absence of any undesirable side-effects—for it must be emphasized at once that fluoride is a powerful poison.

As the years passed, and as the progress reports of the sponsors of these schemes were issued, the results of the experiment proved so encouraging that many other communities indulged in artificial fluoridation of their water supplies, even though the Newburgh-Kingston experiment was far from complete. Dr. Sidney B. Finn, who showed me the whole plan in action at Newburgh-Kingston, told me that he doubted that the control city of Kingston would agree to finish the experiment (it had been designed to cover a ten-year period), because the people of Kingston were disinclined to permit a continuance of the high incidence of caries among their children while the children of Newburgh enjoyed a relatively high degree of immunity from this disease.

At present there are some 225 communities in North America practising fluoridation of water, and many more (some very large and important, like Washington, District of Columbia, Philadelphia, Baltimore, San Francisco) propose to do so, as soon as arrangements can be completed.

Harmlessness.—There are two opposing views on the safety of the practice. Most observers agree that there is no worthwhile evidence of the immediate or remote appearance of toxic manifestations, except (in a minority of subjects) a negligible amount of mottled enamel, so slight as to require careful examination for its detection. The opposite viewpoint has been held by such observers as Spira, who lists some 32 distinct abnormalities (cited by Cox, 1951) as due to the toxic effect of fluorine. There are good reasons for discounting his claims. Cox has discussed the question of toxicity (Cox, 1951) and states that the occurrence in uncontrolled circumstances of toxic manifestations is no reason for denying the benefits of fluorine to very large numbers of urban people when engineers and chemists can see to it that the fluorine content of the water supply is maintained at a level below that which can cause any of the recognized injuries. Many public bodies in the United States have approved and even recommended the widespread fluoridation of domestic water supplies, for example, the following: (i) the American Medical Association through its Council on Pharmacy and Chemistry and its Council on Foods and Nutrition;[1] (ii) the American Public Health Association; (iii) the State and Territorial Health Officers' Association, United States of America; (iv) the United States Public Health Service (Assistant Surgeon-General, Dr. B. D. Forsyth); (v) the American Dental Association; (vi) the State and Territorial Dental Health Directors, United States of America; (vii) the American Association of Public Health Dentists; (viii) the American Water Works Association; (ix) the National Research Council, United States of America.

Efficacy.—Reports of the anticaries action in both children and adults (Forrest, Parfitt and Bransby, 1951) of naturally fluoridated waters are numerous, and although data from the populations practising artificial fluoridation are as yet incomplete, they show an undeniable lowering of the incidence of dental caries in the experimental groups of children who have been "exposed" to the water since birth. The following statement is made in the report of the American Dental Association (1951):

> The D.M.F. (decayed, missing, filled) rates among permanent teeth of 6 to 12 year old children in Newburgh show a consistent downward trend after four years of fluoridation, whereas the D.M.F. rates in the control city of Kingston show no changes. The reduction after four years of fluoride experience in Newburgh is from 20·6 D.M.F. per 100 permanent teeth to 13·9, or a reduction of 32·5 per cent. The rate in Kingston remained at 20·2 D.M.F. per 100 permanent teeth.
>
> An analysis of the number of children with all of their deciduous cuspids and molars completely caries-free shows that there was an increase of 6·7 per 100 children in Kingston while in Newburgh the increase was 21·3, or more than three times as great (Figure I and II after Faber, 1951.)

Bull (1950, 1952) claims a lowering of the incidence by 60% to 65%, also assessed as a D.M.F. rate.

Practicability.—As Bull (1952) has emphasized, fluoridation of domestic water supplies can be carried out in its entirety through the existing facilities. The practice has been in operation long enough (six years) to prove that mechanisms and controls are accurate and simple. As has been mentioned earlier, I was privileged to have the whole Newburgh-Kingston project demonstrated to me. The automatic feeder for introducing the fluorine salt was an unimpressive piece of apparatus measuring scarcely three feet by two feet by two feet. When purchased it cost approximately $400, but it would probably cost $600 today.[1] The capital outlay, therefore, can hardly be held to be excessive. Control of the concentration of the fluoride is simple, and in Newburgh the ordinary staff at the water supply authority's laboratory required no supplementing to carry out the daily tests. The actual cost was therefore limited to the small initial capital outlay and the cost of the fluorine salt (in this case ordinary sodium fluoride). It is estimated that at the present day the cost per person per year does not exceed 12 to 14 cents. Therefore, Bull's claim that the procedure is eminently practicable seems fully justified.

In any public health measure success will depend in part at least on the attitude of the public at large. The dissemination of information in such a form as to be readily comprehended by the public is therefore highly desirable. If the public cannot be persuaded that the measure is both safe and effective, there will be a tendency to avoid the normal use of the public water supply. This has actually happened in one town in New South Wales, not because of the presence of fluorine in the water, but for other reasons; the information was forthcoming that the public water supplies were used only when no alternative supply was available. In the United States there appears to be little doubt in the minds of the informed section of the public that artificial fluoridation is beneficial, as witness the impatience of the people of Kingston at their continued use as controls in the Newburgh-Kingston experiment. (Admittedly two or three centres have rejected the use of fluoridated domestic water supplies.)

Advantages.—The advantages of water fluoridation are as follows: (i) that the administration of the fluoride, toxic though it may be, can now be rigidly and accurately controlled; (ii) that there is no need for the active

[1] The American Medical Association (1951) makes the following statement: "The Councils are unaware of any evidence that fluoridation of community water supplies up to a concentration of one part per million would lead to structural changes in the bones or to an increase in the incidence of fractures. The only difficulty so far revealed is a possible increase in mottling of the tooth enamel. The available evidence based on thousands of observations indicates that the incidence of mottling of the enamel in children who drink water containing fluoride up to a concentration of one part in a million is minimal and detectable only by careful dental examination. It occurs only in a small percentage of children and is so slight as not to present a problem from the point of view of appearance or strength of the teeth. Evidence of toxicity other than the effect on enamel has not been reported in communities where the water supply has several times this concentration. After considering the evidence available at this time, the Councils believe that the use of drinking water containing up to one part per million of fluoride is safe. However, the use of products which are naturally high in fluoride content, such as bone meal tablets, or of lozenges, dentifrices, or chewing gum to which fluoride has been added, should be avoided where the drinking water has been fluoridated. In places where children are subjected to warm temperatures and consequently drink large amounts of water, a lower concentration of fluoride may be necessary to avoid mottling of the teeth."

[1] See, however, Finke *et alii* (1951), who give the cost of the equipment and installation as $1500.

cooperation of the public; (iii) that the cost of fluoridating water is very small.

Disadvantages.—The disadvantages of water fluoridation include the following: (i) Philosophical considerations as to the right of the State to compel the population to ingest fluoride. (ii) The development of mild degrees of mottling of the enamel in a small proportion of the subjects exposed. (iii) Long-range, cumulative, toxic effects, such as stiff back. This objection is held on the ground that insufficient time has elapsed to enable observers to detect the development of these disabilities following artificial fluoridation—this despite the fact that we have adequate knowledge of the lack of deleterious effects in populations exposed for decades to naturally fluoridated water. (iv) Another objection related to industry, particularly in the preparation of various consumable articles, such as dried or concentrated foodstuffs.

2. Topical Application of Fluoride.

Following the observations that the constant use (from birth onwards) of naturally or artificially fluoridated drinking waters appears to result in the development of teeth which are relatively resistant to dental caries, it was suggested that possibly a similar beneficial effect might ensue from the application to the surfaces of erupted teeth of solutions of fluorides, so that, failing the benefits to be derived from the constant ingestion of fluoridated waters, a belated salvation might be effected by transforming the surface layers of the enamel so that they acquired the caries-resisting properties of the naturally fluoridated enamel. The ideas behind this practice (Cox, 1951) were that the initiation of new caries could be prevented by (i) increasing the fluorine content of the enamel, (ii) decreasing its solubility in organic acids, or (iii) increasing its hardness.

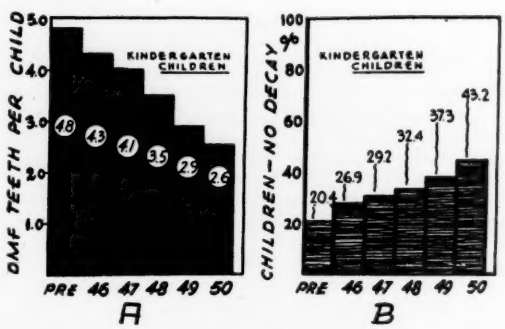

FIGURE I.

A: Sheboygan survey data—D.M.F. teeth per child. B: Sheboygan survey data—children with no decay. (After Faber, 1951.)

Advantages.—There are numerous claims that topical application does indeed effect partial control of dental caries. Earlier results (Knutson, 1949) showed a 40% lowering in the initiation of new cavities. The best results appear to have been obtained on children rather than on adolescents and young adults, who nevertheless do obtain some benefit (Klinkenberg and Bibby, 1950—assessment on D.M.F. rate).

Disadvantages.—The disadvantages of this practice are as follows: (i) the alleged development of various degrees of inflammation of the gums; (ii) the fact that expert operators (dentists and hygienists) are needed to apply a solution of fluoride properly; (iii) the necessity for the cooperation of the patient—that is to say, the public; (iv) the much greater cost as compared with that of fluoridation of water supplies.

MODE OF ACTION OF THE DRUG.

As Cox (1951) points out, the available evidence on the mode of action of fluorides in preventing or limiting dental caries leads to one conclusion—namely, that the mechanism is different for administration through drinking water and for administration by topical application. He makes the following statement:

> It is far better that prevention of dental caries by water fluorides of the order of 1-2 parts per million during the formation of enamel be regarded as a different process than the control of caries in erupted teeth by solutions with 10,000 parts per million fluoride. As long as these processes remain confused by research people the longer the opponents of water fluoridation will offer topical fluorides as a complete equivalent to water fluoridation.

Experiments by Zipkin and McClure (1951) showed that three compounds of fluorine, including sodium fluoride, when administered either orally or intraperitoneally, caused pronounced mottling of the incisor teeth of the rat. The three compounds which caused the mottling produced also a pronounced lowering of the amount of caries if given orally, but none was effective when given intraperitoneally. It is clear that fluoride compounds are not identical in toxicity or metabolism or in effect on the caries incidence, even though used in amounts which are comparable in so far as their fluorine content is concerned (Anonymous, 1952).

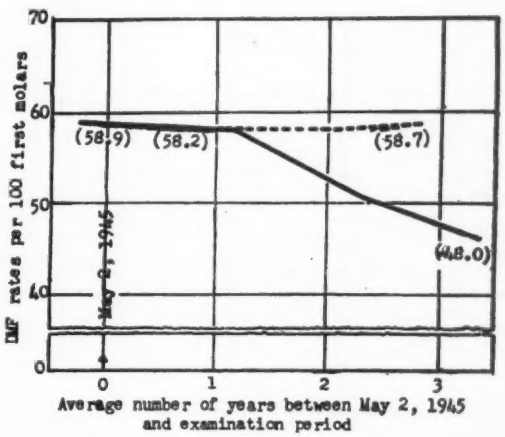

FIGURE II.

Trends in D.M.F. rates of first molars of school children aged six to twelve years. Newburgh, New York (fluoride city), continuous line; Kingston, New York (control city), interrupted line. (Sodium fluoride introduced into Newburgh's water supply on May 2, 1945, as indicated by arrow at "0" years. (After Faber, 1951.)

There are two principal hypotheses to explain the anti-caries action of fluorine. One relates to the properties of fluoridated enamel, the other to the acidogenic activities of oral microorganisms. It is known that in "fluorine" areas there is an increased amount of fluoride in both enamel and dentine and in bone. In these teeth fluorapatite rather than hydroxyapatite (or, in the view of some, carbonate apatite) is the essence of the chemical structure of the enamel. These teeth have surfaces which are harder in the physical sense of being less easy to indent mechanically and harder in the chemical sense of being less soluble in acids. It may well be that the important point here is the almost negligible solubility of certain calcium compounds which contain fluorine—for example, calcium fluoride and fluorapatite.

The other major hypothesis is based on the fact that fluoride is an enzyme poison, so that its presence can limit or even halt the metabolic activities of microorganisms. Cox (1951) suggests that fluoride incorporated in enamel will not be available to poison enzymes until such time as enamel has been dissolved. Of course it is possible that the disruption of minute amounts of enamel will free sufficient fluoride to bring about a retardation or inhibition of enzymic action; thus (Figure III) it is known (Bibby,

1941) that *in vitro* only relatively small concentrations of fluorine (even 1 in 1,000,000) are needed to limit microbial production of lactic acid, and that much greater concentrations are needed to interfere seriously with the rate of growth.

Cox is of the opinion that theories based on the chemical composition of the enamel are not applicable and that the increased physical hardness is more likely to be associated with resistance to the initiation of decay. However, it does not follow that the theoretical bases for topical application are sound because lead fluoride, which on experimental grounds fits the hypotheses better than does sodium fluoride, is not so effective in practical application as is the far more soluble sodium fluoride (Ericsson, 1950[1]). As an

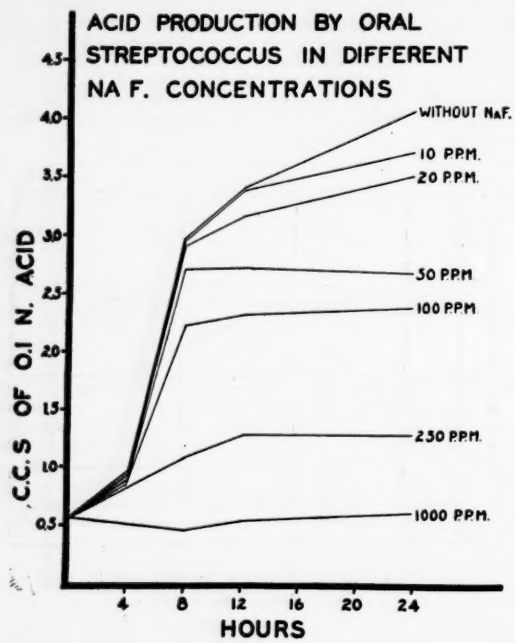

FIGURE III.
After Bibby, 1941.

explanation of the fact that a 1% solution of sodium fluoride is more effective than a saturated solution of lead fluoride in preventing caries, Cox suggests that the sodium fluoride is far more bactericidal. Hence the statement previously made, that in the present state of our knowledge it is far better to regard the prevention of dental caries by fluoridated drinking waters as a different process from the control of caries in erupted teeth by topical application.

THE PRESENT POSITION IN AUSTRALIA.

My interest in this subject dates from the years immediately following Churchill's (1931) demonstration of the existence of fluorine in water responsible for mottled enamel. Attempts to initiate experiments and observations in Australia date from 1947 at the latest, when the Australian Dental Association requested this Institute to furnish a report on the use of fluorine as an anticaries drug. However, because of circumstances beyond our control, it has not been possible either to make preliminary observations or to institute a pilot experiment.

Surveys (Reid and Martin, 1946; Jones, 1949) of domestic water supplies in New South Wales did not reveal any important supply containing one or more parts per million of fluoride. In Queensland, on the other hand, there exist a number of supplies containing one or more parts per million, but none of these serves a large population. It is therefore almost impossible to duplicate the type of observations which have frequently been made in North America and elsewhere, comparing the incidence of caries in fluoride and non-fluoride areas. An attempt is being made to persuade the public health authorities to sponsor the practice of fluoridation in Australia, but it would be a matter for great regret if the present incidence of dental caries was not first determined. While this country is reputed to suffer more than most countries from dental caries, few, if any, figures are available. This absence of information on the incidence of caries in Australia would appear to betoken a lack of interest in this disease, which is paralleled by a similar lack of interest in its prevention by the fluoridation of water supplies. Neither the medical nor the dental profession appears to think either matter important enough for action. However, the medical profession can scarcely escape its share in the responsibility for the dental health of the nation, and it may yet be forced to consider this problem, because, should fluoridation of water supplies be approved,[1] the opinion of the medical profession is bound to be sought on the safety of the procedure.

SUMMARY.

1. Attention is drawn to the claim that fluoridation of domestic water supplies is the most important discovery in public health practice during the last twenty-five years.

2. The history of the relationship of the use of fluoride-containing water to (*a*) the condition known as mottled enamel (chronic endemic dental fluorosis) and (*b*) the incidence of dental caries is sketched.

3. Evidence is adduced in support of the harmlessness, efficacy and practicability of the use of fluoridated domestic water supplies.

4. The principal advantages of this practice are (*a*) the ease of application of the measure to the whole population in a rigidly controlled manner, (*b*) the low cost, and (*c*) the fact that there is no need for the active cooperation of the population.

5. The alleged disadvantages of the practice have no substantial factual support.

6. An alternative method of administering the fluoride, namely, by topical application to the erupted teeth, is briefly discussed, as also are the advantages and disadvantages of this procedure.

7. The mode of action of the fluoride is still uncertain, but is apparently different for the two methods of use.

8. The two most commonly held views are that the incorporation of fluoride into the enamel, whether throughout its thickness (fluoridated water) or in the surface layers only (topical application), (*a*) renders the enamel physically harder and chemically less soluble in the presence of hydrogen ions, and (*b*) limits the acidogenic activities of microorganisms by virtue of the enzyme-inhibiting properties of the fluoride ions.

9. Reference is made to the apparent lack of interest among medical and dental circles in Australia in these preventive measures.

REFERENCES.

AMERICAN DENTAL ASSOCIATION (1951), "Fluoridation in the Prevention of Dental Caries", a Manual by the Council of Dental Health.

AMERICAN MEDICAL ASSOCIATION (1951), "Fluoridation of Water Supplies", Report by its Council on Pharmacy and Chemistry and its Council on Foods and Nutrition, *J.A.M.A.*, 147: 1359.

[1] $Sn^{++}F_2$ and $Pb^{++}F_2$ ions proved *in vitro* to be more effective than NaF in lowering the solubility of enamel. Yet clinically (Bibby, 1947; Galagan and Knutson, 1947) no lowering of the incidence of caries could be obtained by the application of PbF_2.

[1] "The Council, in view of the practice advocated by responsible American medical and dental authorities in respect of fluoridation of water as a factor in the control of dental caries, considers [that] the addition of fluoride to water supplies to be a reasonable and safe measure, provided the addition of this substance is carried out under strict supervision and scientific control to ensure that the percentage does not exceed *accepted* standards laid down by the State Health Departments.

"The use of this substance as a means of self medication is strongly condemned." (National Health and Medical Research Council, Minutes of 33rd Session, 1952.)

ANONYMOUS (1952), "Mechanism of Fluoride Action", *Nutrition Rev.*, 10: 76.
BIBBY, B. G. (1941), "The Bacteriology of Dental Caries", a Symposium on Dental Caries, University of Pennsylvania Bicentennial Conference, Philadelphia: 27.
BIBBY, B. G., DEROCHE, E., and WILKINS, E. (1947), "The Effect of Topical Applications of Lead Fluoride in Dental Caries", *J. Dent. Res.*, 26: 446.
BLACK, G. V., and MCKAY, F. S. (1916), "Mottled Teeth: An Endemic Developmental Imperfection of the Enamel of the Teeth Heretofore Unknown in the Literature of Dentistry", *Dent. Cosmos*, 58: 129.
BULL, F. A. (1950), "Control of Dental Caries by the Addition of Fluorides to Public Water Supplies", *J. Am. Dent. A.*, 41: 146.
BULL, F. A. (1952), "A Public Health Dentist's Viewpoint", *Pub. Health Rep.*, 67: 37.
BULL, F. A. (1952), "A Public Health Dentist's Viewpoint", *J. Am. Dent. A.*, 44: 147.
CHURCHILL, H. V. (1931), "The Occurrences of Fluorides in Some Waters of the United States", *Indust. & Engin. Chem.*, 23: 996. (Cited by Dean, H. T. (1938) in "Dental Science and Dental Art", edited by S. M. Gordon.)
COX, G. J. (1951), "Acute Fluoride Poisoning and Crippling Chronic Fluorosis", The American Association for the Advancement of Science, 118th Meeting, Philadelphia.
DEAN, H. T. (1938), "Endemic Fluorosis and its Relation to Dental Caries", *Pub. Health Rep.*, 53: 204. (This is the first of a series of papers by Dean and his co-workers in this journal.)
EAGER, J. M. (1901), "Denti di Chiae (Chiaie Teeth)", *Pub. Health Rep.*, 16: 2576. (Cited by Dean, H. T., in "Dental Science and Dental Art", edited by S. M. Gordon, Philadelphia.)
ERICSSON, Y. (1950), "Reduction of the Solubility of Enamel Surfaces", *Acta odont. scandinav.*, 9: 60.
FINKE, A. H., and HILDEBRAND, G. J. (1951), "Five Years of Fluoride Operations in Sheboygan, Wisconsin", *Dent. Digest*, 57: 502.
FABER, H. A. (1951), "Developments in Fluoridation—A Summary", *Water and Sewage Works*, 98: 203.
FORREST, J. R., PARFITT, G. J., and BRANSBY, E. R. (1951), "The Incidence of Dental Caries among Adults and Young Children in Three High- and Three Low-Fluorine Areas in England", *Month. Bull. Min. Health*, 10: 104.
GALAGAN, D. J., and KNUTSON, J. W. (1947), "The Effect of Topically Applied Fuorides on Dental Caries Experience", *Pub. Health Rep.*, 62: 1477.
JONES, P. (1949), "The Fluorine Content and Other Chemical Characteristics of Potable Waters in New South Wales", *Dent. J. Australia*, 21: 231.
KLINKENBERG, E., and BIBBY, B. G. (1950), "The Effect of Topical Applications of Fluorides on Dental Caries in Young Adults", *J. Dent. Res.*, 29: 40.
KNUTSON, J. W. (1949), "Evaluation of the Use of Fluorine as a Caries-Control Measure: The Effect of Topically Applied Fluorides", *J. Am. Dent. A.*, 38: 204.
MCKAY, F. S., and BLACK, G. V. (1916), "An Investigation of Mottled Teeth: An Endemic Developmental Imperfection of the Enamel of the Teeth Heretofore Unknown in the Literature of Dentistry", *Dent. Cosmos*, 58: 627.
REID, R. L., and MARTIN, N. D. (1946), "The Distribution of Fluorine in Potable Waters in New South Wales and Tasmania", *M. J. Australia*, 2: 121.
SMITH, M. C., LANZ, E. M., and SMITH, H. V. (1931), "The Cause of Mottled Enamel, a Defect of Human Teeth", University of Arizona, College of Agriculture, Agricultural Experiment Station, Technical Bulletin Number 32. (Cited by Dean, H. T. (1938), in "Dental Science and Dental Art", edited by S. M. Gordon, Philadelphia.)
VELU, H., and BALOZET, L. (1931), "*Darmous (dystrophie dentaire) du mouton et solubilité du principe actif des phosphates naturels qui le provoque*", *Bull. Soc. path. exot.*, 24: 838. (Cited by Dean, H. T. (1938), in "Dental Science and Dental Art", edited by S. M. Gordon, Philadelphia.)
ZIPKIN, I., and MCCLURE, F. J. (1951), "Complex Fulorides. Caries Reduction and Retention of Fluorine in the Bones and Teeth of the White Rat", *J. Dent. Res.*, 30: 524.

THE SIGNIFICANCE OF LYMPHADENOPATHY.[1]

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LYMPH NODES are comprised of specialized aggregations of the reticulo-endothelial cell system, which interrupt the lymph flow at various points. The function of the nodes would appear to be one of filters of the lymph, originating as it does in tissue spaces. This filtering action, one aspect of phagocytosis, is designed to remove from the lymph foreign bodies, whether of foreign protein, bacterial or protozoan origin or cellular disintegration substances.

[1] Read at a meeting of the New South Wales Branch of the British Medical Association at Orange on October 25, 1952.

The action of phagocytosis was appreciated by Aschoff, who differentiated the reticulo-endothelial cell system, grouping together the bone marrow, the liver, the spleen, the lymph nodes and connective tissues for their common property of phagocytosis.

Maximow, appreciating the diverse involvement of the system in disease, elaborated the present concept of the multipotency of the embryonic mesenchymal cell. The reticulo-endothelial cell is widespread throughout the system and capable, when stimulated, of giving rise to lymphatic, fibroplastic, myeloid and erythroid cells as well as the macrophages of the phagocytic system. This concept explains the clinical manifestations of the disordered reticulo-endothelial system in diseases such as lymphadenoma and leuchæmia, and the granulomatous response of the system to viral agents and other agents not yet known.

Thus lymphadenopathy, whilst in many instances a localized expression of phagocytosis, as in acute inflammation, may signify the involvement of the reticulo-endothelial system in some generalized disease process, whether acute or chronic, benign or malignant. The nodes thus mirror, in many instances, widespread involvement of the system.

Anatomically, the node possesses a fibroblastic capsule, penetrated at varying points by incoming lymph channels entering the subcapsular lymph sinus. From the capsule trabeculæ pass into the centre of the gland.

Examination of a section discloses peripherally circular areas of cells (a core of reticulum cells, surrounded by lymphocytes, or masses of lymphocytes) surrounded by freely anastomosing lymph and blood sinusoids—the germinal follicles. In the centre of the gland are cords of reticular fibres, with mesenchymal cells, lymphoblasts, lymphocytes, histiocytes and reticulum cells lying upon them—the medullary portion of the gland. Lymph channels pass from the hilum, where blood vessels enter and leave the gland. The undifferentiated mesenchymal cell, when appropriately stimulated, is by its inherent multipotent character able to produce granulocytes, lymphocytes, fibroblasts, histiocytes *et cetera*, and thus hyperplasia of the node may be due to any of these specialized cellular responses.

This multicellular response is appreciated on examination of sections of the node of Hodgkin's granuloma, but in typical clinical cases of Hodgkin's paragranuloma it is not so evident, and thus may occasion difficulty in the histological diagnosis of this entity. For a similar reason it will be appreciated that in the more malignant diseases of this group a histological labelling is often impossible, and at most times difficult. Robb Smith classified the reticuloses into medullary, follicular and sinus groups, on the anatomical cellular response of the node system to varying disease processes. A medullary hyperplasia was noted in Hodgkin's disease and the metabolic storage diseases such as Gaucher's. A follicular hyperplasia was the principal finding in Brill-Symmers reticulosis, and a sinus hyperplasia was noted as the response to inflammation—whether bacterial, as in enteric fever, protozoan or other—and thus the prime response in phagocytosis.

The role of the node and the reticulo-endothelial system in phagocytosis has been the subject of much original work in the past decade. Wan and Jung in their studies with thorium dioxide ("Thorotrast") demonstrated that the amœboid cells of the system avidly absorbed "Thorotrast", and frequently fused with each other to form giant cells in this process.

McKenny and Mellon, in studies of sulphanilamide-treated streptococcal peritonitis in mice, considered that some strains of bacteria were phagocytosed by neutrophile cells solely, but others appeared to require the presence of macrophages for their removal.

Baker and Entiknoff demonstrated the actual engulfment of bacteria in lymph nodes of the appendix. In this work it is appreciated that granulocytes, serum opsonins *et cetera*, play a major role in phagocytosis.

Spies demonstrated a curious phenomenon, that in leuchæmia the phagocytic response of the granulocytes is poorer than in normal animals, whilst in anæmia this response is greater. In malnourished animals the phagocytic response is lessened, and reinforcement of the normal defence process by the reticulo-endothelial system may account for the adenopathy frequently noted in this state.

Thorne and others have stressed the depression of the reticulo-endothelial system with ACTH and cortisone therapy, and there is little doubt that the adrenal cortex exerts a regulatory control on the functional activity of macrophages and on the system as a whole. Whilst the depression is of temporary nature, the clinical application is occasionally of value, as in the following case record.

Mrs. B. was admitted to Sydney Hospital in acute respiratory distress. A diagnosis of reticulum cell sarcoma was established, but deep X-ray or nitrogen mustard therapy was considered inadvisable in view of the severe respiratory embarrassment. Cortisone in full dosage was administered, and in five days the reduction in size of the mass was sufficient for the administration of deep X-ray therapy.

The temporary nature of the response to cortisone in the reticuloses is appreciated.

It is notable that whilst the prognosis of these diseases is unaltered by deep X-ray or kindred therapies, remissions occur characterized by hæmoglobin elevation, a normal leucocyte response, reduction in size of glands, spleen and liver, symptomatic improvement and objectively a gain in weight.

In that group of diseases known as "the collagen diseases", lymphadenopathy is noted, especially in *lupus erythematosus* and dermatomyositis, and to a lesser extent in rheumatoid arthritis and serum sickness. Excluding serum sickness, the ætiology of the members of the group is obscure, and a common link in these diseases is their response to cortisone.

Lymphadenopathy in Acute Infectious Disease.

The acute infectious fevers present clinical patterns that enable a diagnosis to be made readily. However, some acute lymphadenopathies present difficulty at times in diagnosis. Upper respiratory tract infections, acute infectious mononucleosis and acute infective hepatitis may present with confusing clinical features.

The American Army Commission on Acute Respiratory Diseases excluded such readily appreciated entities as lobar pneumonia, and devoted its attention to the three clinical entities, atypical pneumonia, acute tonsillitis and pharyngitis and undifferentiated acute respiratory disease. Atypical pneumonia is rarely associated with lymphadenopathy. Of the remaining diseases, acute tonsillitis and pharyngitis, which comprised less than 25% of upper respiratory infections, were further subdivided into three entities: (i) non-streptococcal infections of unknown ætiology (50%); (ii) true streptococcal infections (25%); (iii) streptococcal infections, but without clinically estimable antibody (25%). This third group was regarded as of streptococcal origin.

Adenopathy was commonest in the streptococcal group. The undifferentiated acute respiratory disease group was the commonest clinical entity. In this group the condition was characterized by a typical clinical picture. Headache and malaise were usually of brief duration, but a fever of 99° to 101° F. was commonly noted. The significant feature of the illness was the subsequent development of cough, hoarseness and subcostal or chest pain, with a tendency for cough and sputum to persist.

The Commission was of the opinion that this disease was of viral origin, a finding further suggested by the normal leucocyte count. Non-streptococcal pharyngitis and tonsillitis was again considered as of viral origin, this entity being characterized by injected fauces, follicular hyperplasia and cervical adenopathy. The symptomatology common to both diseases was the persistent lower respiratory tract involvement. Adenopathy was commoner in the latter group than in the former. A very similar clinical picture has been noted in epidemic form in army camps during the past winter, and in this disease difficulty was frequently encountered in the exclusion of infectious mononucleosis.

Infectious mononucleosis may present considerable difficulty in diagnosis. In epidemics the cervical glandular enlargement is the common presentation; but unusual cases of central nervous system, hepatic or renal involvement may occur, in which the diagnosis is not at first considered.

Diverse manifestations, such as a generalized febrile and toxic state, in which hepatic and renal failure are the presenting phenomena, may precede lymphadenopathy by days, or again the lymphadenopathy may be of minimal degree and its significance not appreciated. Also unusual complications of the disease, spontaneous rupture of the spleen, hepatic involvement, myocarditis and central nervous system involvement, may be of severe degree. Histologically there may be focal areas of hepatitis. Even the blood picture may present difficulties in diagnosis, a similar picture being noted in pertussis, rubella, varicella and mumps, and it is notable that in some cases the characteristic blood picture may be a relatively late occurrence. Again difficulty may occasionally be experienced from the apparent blood picture of acute monocytic leuchæmia.

Bender is of the opinion that a diagnostic titre of heterophile antibodies occurs in the second week of the disease and is of the order of 1:56. The importance of a rising titre in diagnosis need not be stressed.

Infectious hepatitis is again a disease that involves the reticulo-endothelial cell system, and adenopathy is usually present. The presence of adenopathy in this disease may be of clinical value in the differential diagnosis of jaundice, and especially so in the exclusion of obstructive jaundice from the differential diagnosis.

The Paul-Bunnell test may be the sole differentiating agent in diagnosis of infectious mononucleosis with severe hepatic involvement from infectious hepatitis. However, in most instances of this type, hepatic needle biopsy is a useful differentiating agent. The natural history of both diseases is remarkably similar. Both diseases have, commonly, a good prognosis. Severe hepatic involvement, central nervous system involvement and myocarditis usually portend a grave outcome; and especially in infectious hepatitis, whilst the liver bears the brunt of the infection, the involvement of the whole system should be remembered.

Dougherty demonstrated the ingestion of particulate bodies by microglia and suggested that these cells were part of the reticulo-endothelial system. Lymphadenopathy, prominent in mononucleosis, less prominent in infectious hepatitis, is but one expression of a widespread reticulo-endothelial system involvement.

The Chronic Lymphadenopathies.

Of the chronic lymphadenopathies, sarcoidosis presents many difficulties in diagnosis. This disease, a chronic infection having a predilection for the reticulo-endothelial system, is characterized by clinical progression and regression. Localized and generalized forms of the disease occur, and any tissue from skin to bone may be the site of the disease. The skin manifestations, the nodular sarcoids of Boeck, the subcutaneous sarcoids of Darrier, the uveo-parotid syndrome, the pre-auricular and post-auricular adenopthay, the osseous manifestations (circumscribed cystic areas of rarefaction or the diffuse form of multiple areas of rarefaction in the metacarpal and metatarsal bones), the normal leucocyte count or leucopenia, the mild monocytosis, the biochemical findings (elevated calcium and protein levels), and the pulmonary involvement are clinical features suggesting the diagnosis.

However, the composite clinical picture is uncommon, the disease presenting as an isolated lesion of an organ with or without adenopathy, and recourse may be made to splenic, tonsillar or hepatic biopsy for confirmation of the diagnosis.

Many criteria for a diagnosis of this disease are common to other granulomatous processes. Thus monocytosis is accepted as evidence of reticulo-endothelial system activity, and is the most common hæmatological finding in lymphadenoma, as well as in the granulomatous processes. Brucellosis may present with pulmonary involvement, iridocyclitis and similar bony changes. Rabello described a similar adenopathy, clinically and histologically, and tuberculin anergy in leprosy. Peripheral generalized lymphadenopathy is uncommon in tuberculosis. Acute miliary tuberculosis is confirmed radiographically, by the clinical findings of acute toxæmia and by the temporary response to streptomycin. In subacute forms, an obscure fever, peripheral adenopathy involving the preauricular, cervical, axillary, inguinal and epitrochlear regions with evidence of upper mediastinal glandular enlargement, a palpable spleen, and occasional uro-genital involvement or pleural effusion or ascites are the common manifestations.

Amberson is of the opinion that death may occur in three to six months in this group, but that some survive for longer periods. He regards the response to streptomycin as more lasting in this group. In the chronic form of this disease, a chronic pulmonary focus of infection is usually radiographically noted, and peripheral adenopathy more commonly demonstrates calcification in the nodes. The distinguishing feature of sarcoidosis from these forms of generalized tuberculous lymphadenopathy is the regression and progression of the sarcoid lesion.

Discrete localized tuberculous glands are secondary to a tuberculous focus, or are sequelæ of the bacteriæmic phase. Caseation and necrosis finally produce the classical inflammatory manifestations. Kala azar may again present a similar picture to sarcoidosis. Histoplasmosis, with its world-wide distribution and predilection for the adult male and children, may demonstrate granulomatous lesions, from which the organism may be absent.

A newer occupational disease, beryllosis in its generalized form, may present a similar clinical picture to sarcoidosis. One feature common to these varied disease is their interrelationship to the reticulo-endothelial system. It is not surprising, therefore, that the typical sarcoid histologically bears such a close relationship to these varied granulomatous processes that even when it is classical, histological diagnosis is not proof alone for the diagnosis of sarcoidosis.

The reticulo-endothelioses present a varied and diverse clinical picture complicated by naturally occurring remissions. The natural history of these diseases is usually one of brief duration. Exceptions due to prolonged remissions occur notably, in Wintrobe's histological classification, in the paragranuloma of Hodgkin—the isolated glandular finding in the neck and in the mediastinum. Survival rates of fifteen to twenty-one years are on record in this group. His classification of these tumours is into two groups on a histological basis: (i) with a relatively simple pattern —lymphosarcoma, reticulum cell sarcoma; (ii) with a more complex pattern, for example, Hodgkin's disease.

His conception of Hodgkin's disease is based on essentially three types—the paragranuloma, the granuloma and the sarcoma groups—and is notable for his appreciation of the potential merging of any one type into the other types on a histological basis.

It is notable again that whilst this classification is essentially a histological one, clinical features of adenopathy presenting are an important factor in his classification. This factor of the transition of any one type of Hodgkin's disease into the other groups is not a feature peculiar to Hodgkin's disease, but is common to all histologically diagnosed diseases of this group. Thus numerous examples occur in the literature in which biopsies from a single patient at varied sites and at subsequent times showed the histological features of all members of the group—for example, reticulum cell sarcoma, Hodgkin's lymphosarcoma, fibrosarcoma and lymphatic leuchæmia. The classification of these diseases on a histological basis is thus an artificial one and of limited value in prognosis.

The clinical evolution of the disease and its response to therapy are probably the best guide to prognosis in the reticulo-endothelioses and the best basis for a classification.

The clinical features of the adenopathy are common to all the reticulo-endothelioses, the prognosis being more grave in those cases in which the glands are subjectively painful or tender on palpation and when generalized adenopathy is present. Visceral involvement, such as hepatic involvement, is of grave omen and denotes that the response to therapy will be of brief duration or unsatisfactory.

The individual members of this group tend to occur at various decades of life, but even this factor in diagnosis is offset by the occurrence of Hodgkin's disease from the first to the eighth decade. The clinical presentation of these diseases may be by glandular enlargement, visceral involvement, cutaneous manifestations, such as *mycosis fungoides*, and pyrexia of unknown origin, to mention but a few of the commoner modes of presentation. Thus the natural history of the reticuloses follows no one clinical pattern, but is notable in each case for bizarre and unusual features.

The hæmatological findings, leucopenia, leucocytosis or normal cell count, with lymphocytopenia and monocytosis, are common to all and, apart from the usual occurrence of anæmia, of little value in diagnosis. However, a leucocytosis may denote visceral involvement, whilst a leucopenia of severe degree may denote bone-marrow involvement of severe degree. A rapidly falling hæmoglobin level is of grave prognosis.

The histological confirmation of the diagnosis of reticulosis is essential in view of the gravity of the prognosis for the group, but the histological diagnosis should be accepted in its generic sense. The prognosis is based essentially on the clinical appraisal of the patient—adenopathy, extent of visceral involvement and rapidity of evolution of the disease, and response to therapy.

The following case histories are mentioned to stress the unusual and prolonged feature of obscure pyrexia.

R.B., aged thirty-one years, presented on August 16, 1951, with a history of feverish attacks (100° to 101° F.) lasting for five to ten days, for which such diagnoses, unsatisfactory clinically, were established as gastritis and fibrositis, over a period of eight years prior to his developing a continuous fever with severe night sweats and without objective physical findings. This fever persisted for two months prior to the development of *mycosis fungoides* and peripheral lymphadenopathy. The course of this illness was marked by hepatic and splenic enlargement and progressive normocytic anæmia with ultimately granulocytopenia. The response to treatment with nitrogen mustard and cortisone was minimal.

L.V., aged fifty-one years, presented at the Repatriation General Hospital, Concord, with a classical Hodgkin's paragranuloma, clinically and histologically confirmed. He had two series of treatments with irradiation, and eighteen months later again presented with an indurated violacious lesion measuring about three inches by two inches on the right arm. Response to irradiation was minimal, and excision of the tumour was performed. A histological diagnosis of fibrosarcoma was made. This man has shown no further adenopathy or visceral involvement in the six months subsequent to operation.

Y.P., aged fifty-three years, was admitted to Sydney Hospital on January 11, 1949, for an obscure fever. The history was significant for a mediastinal "cyst" which had been noted in 1940. On examination of the patient, hepatomegaly and splenomegaly were found to be present, with adenopathy suggestive of a reticulosis. A blood count on his admission to hospital revealed between 3,000,000 and 4,000,000 erythrocytes per cubic millimetre, a hæmoglobin value of 10·7 grammes *per centum*, and 9050 leucocytes per cubic millimetre, made up of neutrophile cells 12%, lymphocytes 82% and monocytes 4%. A diagnosis of leuco-erythroblastic anæmia was made. Seven days after his admission to hospital a further blood count disclosed a hæmoglobin value of 10·7 grammes *per centum* and a total of 57,000 leucocytes per cubic millimetre, 88% being lymphocytes. A sternal biopsy confirmed a diagnosis of lymphatic leuchæmia.

Reticulo-endothelioses presenting as lymphatic leuchæmia have been described by many authorities, notably Willis, and the evolution of this disease suggests this diagnosis. It was notable in this case that comparative X-ray examinations of the mediastinum disclosed a great increase in the

size of the mass on the patient's admission to hospital, as compared with the earlier X-ray findings in 1940.

Wintrobe is of the opinion that extracutaneous structures are rarely involved in *mycosis fungoides*, but this has not been my experience, this presentation being notable for the brief clinical response to nitrogen mustard or irradiation therapy and the rapid evolution of the disease, tending to involve all tissues of the reticulo-endothelial cell system.

The following case histories demonstrate the minimal response to therapy and the involvement of extracutaneous tissue.

A.B. presented at Sydney Hospital in 1951 with *mycosis fungoides* of three months' duration. The response to deep X-ray therapy was disappointing and the course of the disease was marked by the occurrence of lymphadenopathy, splenomegaly, acute toxæmia and anæmia. The response to nitrogen mustard therapy was again minimal.

A further case of *mycosis fungoides*, the patient, S.T., being aged fifty years, who was admitted to Sydney Hospital in 1952, has shown a similar clinical evolution, with again a failure to respond to therapy.

In conclusion, the reticulo-endothelioses may present in any bizarre way and at any site where reticulo-endothelial tissue occurs. A diagnosis of reticulo-endotheliosis, primarily clinical, but confirmed histologically, denotes a grave prognosis, the prognosis being gauged by the clinical history and findings and response to therapy. A histological diagnosis is in itself of little value in determining the type of therapy required; such therapy—irradiation or nitrogen mustard—is primarily decided by the extent of involvement of the tissues by the disease.

The classification of the members of this group into clinical entities, if attempted, is better made on clinical than on histological grounds.

Leuchæmia in all its forms is finally diagnosed from the peripheral blood picture or bone marrow findings. Lymphadenopathy is minimal in the myeloid group and common in the lymphoid. The discrete, non-tender, firm glands with their predilection for the cervical chains, and in the generalized form the involvement of the pre-auricular and post-auricular, epitrochlear, axillary and inguinal sites, with splenomegaly, readily suggest the diagnosis. In the acute forms, the rapidly ensuing anæmia, the bleeding tendency, the oral findings with tributary cervical adenopathy, and the blood picture suggest the diagnosis. The prognosis is grave, and there is no record in the literature of natural remissions prolonging life beyond twelve months from the time of diagnosis. The reports of Dameshek and others of survival rates of thirteen to twenty-one months following the administration of aminopterin and folic acid therapy are most encouraging. Regional lymphadenopathy has been found in agranulocytosis and refractory anæmia, but it is not a prominent feature of these diseases.

Cancer research has stimulated interest in regional lymph node metastasis, and the mechanism of metastasis, as now accepted, is primarily by the venous system. However, whilst lymphatic permeation as the usual means of dissemination is not now accepted, lymphatic embolism from the primary site to the regional node may obstruct the flow of lymph and cause collateral channels to disseminate the tumour to neighbouring nodes.

This factor is an influence in the present surgical trend of more radical technique, and is evoking a keener appreciation of the indications and contraindications for irradiation and the use of radioactive therapy.

Lymphadenopathy in children in the absence of obvious disease processes may present difficulties. However, this is the period of life associated with the acquisition of antibodies, and this glandular hypertrophy, often with tonsillar hypertrophy, is but another expression of widespread reticulo-endothelial activity, and a physiological evidence of the mechanisms of defence. Lymphoid tissue is prolific in the young and is labile, and thus care should be exercised in such procedures as tonsillectomy indicated solely by lymphoid hyperplasia.

THE SIGNIFICANCE OF GENERALIZED LYMPH NODE ENLARGEMENT.[1]

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LYMPH NODE ENLARGEMENT occurs in many diseases, especially those due to infections. However, in this paper it is intended to review lymph node enlargement as a manifestation of primary lymphoid disorder mainly in cases seen at autopsy.

The chief lymphoid diseases are (*a*) follicular lymphoblastoma, (*b*) lymphosarcoma, (*c*) Hodgkin's disease, and (*d*) lymphatic leuchæmia.

Follicular Lymphoblastoma.

Follicular lymphoblastoma, which is also known by the terms "giant follicular lymphadenopathy" and "Brill-Symmers disease", is a pre-malignant hyperplasia of lymph nodes which may terminate in lymphosarcoma, polymorphic reticulosarcoma (Hodgkin's sarcoma), Hodgkin's disease or leuchæmia. The peak incidence is in the fifth decade, but no age group is exempt.

The enlargement of lymph nodes may be generalized, but more commonly it is confined to one or more groups of nodes, such as the cervical, axillary, inguinal or abdominal groups. The nodes are discrete and firm, measuring up to five centimetres in diameter, but in the average case the enlargement is much less than this. Splenic enlargement occurs in 20% of cases.

Wherever there is lymphoid tissue, there may be follicles of this type present, and there are cases recorded of the intestine and appendix being the primary involved sites.

When malignant change supervenes (leuchæmia is included for the purposes of this paper amongst the malignant diseases), the cells of the abnormal follicles in the nodes infiltrate the surrounding lymphoid tissue.

The essential lesion in this form of lymphadenopathy is a multicentric hyperplasia of primitive lymphoid cells forming follicular groups in preformed lymphoid tissue.

In most cases there are no symptoms until there has been a change to one of the more lethal diseases.

In three cases at Royal Prince Alfred Hospital there has been intestinal obstruction due to lymphosarcoma of the abdominal lymph nodes, while the superficial nodes still displayed the pre-malignant condition of follicular lymphoblastoma.

Follicular lymphoblastoma may be accompanied by the following other conditions:

1. Generalized skin conditions, such as erythrodermia, eczema or exfoliative dermatitis. It is noted here that generalized lymph node enlargement may occur in erythrodermia and exfoliative conditions due to other causes, and in these it is a form of reactive hyperplasia.

2. Spiegler-Fendt sarcoid. Brownish nodules and infiltrations appear in the face, sometimes the chest and elsewhere. They are composed of lymphoid tissue showing the same follicular lymphoblastomatous appearances.

3. *Mycosis fungoides.* This usually occurs primarily as a skin disorder, but may be associated with follicular lymphoblastoma. It is a multicentric proliferation of reticulum cells occurring in the skin. Its behaviour parallels that of follicular lymphoblastoma, progressing to one of the more fatal lymphoid disorders after a variable and usually lengthy period.

Prognosis.

The natural course of the disorder is to a fatal termination.

[1] Read at a meeting of the New South Wales Branch of the British Medical Association at Orange on October 25, 1952.

The shortest period between onset and death from sarcomatous transformation amongst the Royal Prince Alfred Hospital cases was in the case of a woman of forty-five years who had cervical enlargement of lymph nodes for six months. Biopsy revealed follicular lymphoblastoma. Despite radiotherapy she progressed to polymorphic reticulosarcoma and died eleven months later.

On the other hand a woman of fifty-three years complained of lumps in the posterior cervical triangle of six months' duration. A biopsy revealed follicular lymphoblastoma. She was treated by irradiation; and now, ten years afterwards, she has further enlarged lymph nodes in the axilla, and these now show transformation to lymphosarcoma.

While the lymph nodes involved in this disorder are very sensitive to irradiation, recurrence usually takes place. The condition is frequently located in one or more groups of accessible lymph nodes, and irradiation may protect the patient for a period up to twenty years.

Of 34 patients at Royal Prince Alfred Hospital in the period 1940-1950, five only appear to be perfectly well. After irradiation these are free from lymphadenopathy for two, two, two, four and ten years, although one has a persistently enlarged spleen.

Four of the patients are untraced, and of the remainder 11 are dead. The other 14 all have clinical or histological evidence of lymphosarcoma.

The causes of death were as follows: cardiac failure without malignant transformation, 1; pulmonary tuberculosis without malignant transformation, 1; polymorphic reticulosarcoma, 4; lymphosarcoma, 4; Hodgkin's disease, 1.

Lymphosarcoma.

Lymphosarcoma is a true malignant neoplasm, which often commences at one site, where it persists for a variable, though not prolonged, period, after which multicentric foci of lymphosarcoma appear in other regional and distant lymph nodes as well as the spleen, liver and kidneys. Sometimes malignant change appears in many centres initially.

In a number of cases, as already mentioned, the transformation of follicular lymphoblastoma can be observed. In some the histological appearance suggests that follicular lymphoblastoma has been present. In the majority, however, the histological appearance does not give any indication as to whether all cases commence in such a manner.

That lymphosarcoma is a true malignant neoplasm is evidenced by its invasive nature, destroying whatever tissue obstructs its pathway. This is different from the infiltrations of leuchæmia, for example.

Lymphosarcoma has several histological forms depending upon the degree of differentiation of the reticulum cells which are the originators of the whole process. Classification according to histology does not help the clinicians, as the behaviour of the various neoplasms is clinically identical.

In 6500 consecutive autopsies at Royal Prince Alfred Hospital there have been 32 examples of lymphosarcoma; three-quarters of the subjects affected were over forty years of age. An attempt to define the primary growths has been as follows: abdominal lymph nodes, 13; mediastinal lymph nodes, 8; intestinal tract, 3; spleen, 2; testis, 2; cervical lymph nodes, 1; bone, 1; mouth, 1; dura, 1.

From these figures lymphosarcoma is obviously a disorder in which the deeper structures of the body are predominantly or primarily involved. Superficial lymph node involvement alone is rare, but does occasionally occur. This type could possibly be relieved by radical surgery in the early stages. Cure occasionally results, as in the following case:

A man, aged forty-six years, had an enlarged tonsil which was removed in 1930. This was thought to have an appearance suggestive of lymphosarcoma. A further growth appeared in the upper part of the tonsillar fossa, and three months after the tonsillectomy it was more than one inch in diameter. This was widely excised, and sections of it show the appearances of polymorphic reticulosarcoma. The man is still well and clinically free of cancer in 1952.

Symmetrical involvement of organs is sometimes, though rarely, a feature of lymphosarcoma; the kidneys, testes, breasts and suprarenal glands are the primary or predominant sites. Mikulicz's syndrome, when caused by lymphosarcoma, is a good example of this symmetry.

Lymphosarcoma in humans can be divided roughly into the following groups in order of frequency: (i) Involvement of lymphoid tissues of the abdomen, including intestines and retroperitoneal lymph nodes. (ii) Involvement of mediastinal lymph nodes with invasion of adjacent structures, such as the pericardium, heart and lungs and pleura. (iii) Involvement of superficial lymphoid structures including the skin and integumentary tissues. (iv) Involvement of paired organs. (v) Lymphosarcomatous tumours which are accompanied by lymphatic leuchæmia as a terminal manifestation.

The only groups in which curative measures may be successful are those in which the superficial lymph nodes alone are involved and occasionally when a single organ such as the spleen is the seat of disease.

Occasional cases of intestinal obstruction are due to lymphosarcoma of the bowel. In these, the prognosis is often relatively good, as in a high proportion the disease is confined to a segment of gut which can be resected, though there is the possibility of recurrence in regional lymph nodes.

When there is reasonable evidence of lymphosarcoma being localized to one group of lymph nodes, or one organ, radical surgery should be attempted. The few published reports of radical surgery followed by irradiation are very encouraging.

Hodgkin's Disease.

Hodgkin's disease is a disorder of the lymphoid system of unknown ætiology. It has some of the characteristics of neoplasia and some of inflammation. It behaves like a malignant neoplasm in that there are newgrowths which will kill the patient eventually, yet it does not metastasize.

It is manifested by enlargement of various groups of lymph nodes and involvement of certain viscera, most commonly the spleen. It mostly occurs in the decade twenty-one to thirty years, but may occur at any age. Twice as many males as females are affected.

Microanatomy.

The architecture of the involved tissues, whether in lymph node, spleen or other organ, is destroyed, and the parenchyma is replaced by fibrosis with varying numbers of cells. These cells are proliferating reticulum cells (Reed-Sternberg cells are multinucleate reticulum cells) and blood cells derived from these reticulum cells, namely, megakaryocytes, lymphocytes, plasma cells and granulocytes, including eosinophile leucocytes. Sometimes necrosis occurs.

Macroscopic Anatomy.

The lymphoid tissue of the body may be divided into three main groups as far as Hodgkin's disease is concerned: (i) The cervical, axillary, thoracic, abdominal and inguinal lymph nodes, which bear the brunt of this disorder. (ii) The spleen, liver, skin, kidneys *et cetera*, which are less frequently involved. (iii) Submucosal lymph nodes of the alimentary tract and pharynx, which almost invariably escape.

In the 25 cases at Royal Prince Alfred Hospital which have been investigated at autopsy in the past twelve years, the superficial lymph nodes have been only the minor manifestation. While the cervical lymph nodes are usually the first to call clinical attention to the possibility of Hodgkin's disease, they merely indicate that there is involvement of the deeper lymph nodes, and so are a late sign.

Of the 25 subjects, 21 had cervical lymph node enlargement.

Involvement of axillary lymph nodes almost invariably means larger masses of involved lymph nodes in the mediastinum, even if there is no X-ray confirmation of this. Similarly, involvement of the inguinal lymph nodes means that there are large masses of involved abdominal lymph nodes. These findings are the result not only of the Royal Prince Alfred Hospital investigations, but also of the studies of Symmers amongst the necropsies at Bellevue Hospital.

Complications.

Sometimes the complications of Hodgkin's disease dominate the clinical picture.

Hæmolytic anæmia, associated with normal cervical lymph nodes or slightly enlarged superficial lymph nodes, may be misleading. However, a careful clinical examination usually, though not always, reveals a superficial lymph node which may give the diagnosis to the pathologist. In such cases, also, liver biopsy may prove helpful. It is often involved in patients with severe anæmia. In one such case at Royal Prince Alfred Hospital there was no superficial lymph node involvement, but at necropsy there was extensive liver and splenic disease.

Fever, associated with enlarged lymph nodes, is a not uncommon finding in a number of disorders, and when a diagnosis is not found after the usual laboratory tests, lymph node biopsy is imperative.

Tuberculosis as a complication was observed in three of the 25 autopsies.

Paraplegia was the presenting symptom in one case.

Treatment.

In Hodgkin's disease there is widespread lymphoid involvement. Treatment in such cases must be of a general type. In a few cases the disorder remains localized for a considerable time, and occasional patients appear to have had their lives prolonged by surgery at this stage followed by irradiation.

Lymphatic Leuchæmia.

Amongst the 6500 autopsies performed at Royal Prince Alfred Hospital in the last twelve years, there have been found 32 examples of lymphatic leuchæmia. Five were of aleuchæmic lymphatic myelosis. Six were of acute lymphatic leuchæmia occurring in patients aged twenty-two months, and two, four, four, fourteen and sixteen years. Twenty-one were of chronic lymphatic leuchæmia, all except four being in patients over fifty years old, with the majority in the seventh decade.

Autopsy Findings.

There was splenic enlargement in all chronic cases, and in all except one there was enlargement of the superficial lymph nodes. The largest spleen weighed 3029 grammes, and the smallest 230 grammes.

Lymph Nodes.—The lymph nodes of both acute and chronic lymphatic leuchæmia are almost always enlarged. They are discrete but numerous. The average enlargement is only to about twice or three times the normal size. In two of the acute cases and in one of the chronic cases there was no enlargement of the superficial lymph nodes. However, superficial lymphadenomegaly is fairly constant. Deeper lymph nodes are less often involved than the superficial nodes but may be much larger. The largest one encountered in the Royal Prince Alfred series was a mesenteric lymph node the size of an orange.

Spleen.—The spleen was enlarged in every case except one of aleuchæmic lymphatic myelosis. In the others the smallest spleen was one and a half times normal, and the largest was 25 times normal (over 3000 grammes).

Liver.—Enlargement of the liver is fairly common also.

Bone Marrow.—In all cases in which the marrow was examined, it was densely infiltrated by lymphocytes.

Blood.—The total leucocyte count varied from about twelve thousand to several hundred thousand per cubic millimetre and the predominant cell was the immature lymphocyte.

Diagnosis.

In practice, the diagnosis is by examination of the blood.

In doubtful cases, when the blood count is not high, and in aleuchæmic cases, enlarged lymph nodes, splenomegaly, hepatomegaly and fever may be the chief features. Diagnosis can be rapidly established by bone marrow biopsy.

The examination of a lymph node is not always reliable, in that a lymph node may be selected which is normal, or one may be selected which is so heavily involved that it is impossible to distinguish it from lymphosarcoma.

Leucocytosis is not necessary for suspecting leuchæmia, and a patient with lymph node enlargement and splenomegaly must be investigated with this diagnosis in mind.

Treatment.

Lymphatic leuchæmia being a generalized reticulo-endothelial hyperplasia, effective treatment must be general too.

Aleuchæmic Myelosis.

Aleuchæmic myelosis is a rare condition. It is frequently diagnosed, but those cases to which this name is given are often examples of leuchæmia in which abnormal cells are present in the peripheral blood though the total leucocyte count is not greatly increased.

In aleuchæmic lymphatic myelosis, there is infiltration of organs as in leuchæmia. The bone marrow is leuchæmic, but the peripheral blood picture is normal.

On one such case, a young man of twenty-five years presented with the complaint of cervical lymph node enlargement and fever of six weeks' duration. There was hepatomegaly but no splenomegaly, and all the superficial lymph nodes were slightly enlarged. The blood count was normal. The patient was treated with nitrogen mustards, but he died. The total duration of his illness was three months. At autopsy he was found to have slight generalized lymph node enlargement and very heavy infiltration of both kidneys and the liver.

There have been five such cases at Royal Prince Alfred Hospital amongst the last 6500 autopsy cases. Four of the subjects were males, and their ages have been ten years, twenty-five years, fifty-six years and fifty-seven years. The female subject was five years old.

The condition is therefore rare. Lymph node enlargement is almost invariably present.

Diagnosis is by lymph node biopsy or, preferably, marrow biopsy.

Lymph Node Biopsy.

It is desirable to select a group of lymph nodes for biopsy, rather than a single node, as it is quite possible to find unaffected lymph nodes in advanced cases of lymphoid disease.

If a representative lymph node is examined and the histological appearances are only those of reactive hyperplasia, it is a fairly safe assumption that the patient is not suffering from a primary lymphoid disorder.

Except in young children, the inguinal lymph nodes are not usually very satisfactory, as they frequently show enlargement and fibrosis due to chronic infections of the feet.

Summary.

Follicular lymphoblastoma, lymphosarcoma, lymphatic leukosis and Hodgkin's disease are primary lymphoid disorders. To these may be added *mycosis fungoides*, which behaves in a fashion comparable to that of follicular lymphoblastoma.

Apart from some cases of follicular lymphoblastoma and *mycosis fungoides* in which death is due to other causes, the primary lymphoid disorders are almost uniformly and invariably fatal. In the majority of cases there is enlargement of superficial lymph nodes, and diagnosis can be readily established by lymph node biopsy.

THE TREATMENT OF PEPTIC ULCERATION.[1]

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ANY discussion on peptic ulceration must take cognizance of the recent accumulation of knowledge concerning its geographical peculiarities, age and sex incidence, and frequency of occurrence in different walks of life. In the United Kingdom it has been shown that the professional classes and those burdened with responsibility are the heaviest sufferers, whilst agricultural workers show the lowest incidence. Contrary to expectations, shift workers suffer no worse than their more regular comrades. The gradual but relentless march or progress of duodenal ulceration has been noted in most countries the world over.

The belief that gastric and duodenal ulcers are distinct as regards their ætiology and behaviour has gained considerable support during the past decade.

Duodenal ulcers occur essentially in well-nourished young men, equal numbers coming from all social classes. The clinical story is usually so characteristic that the radiological confirmation should be of secondary importance. The clinical pathologist finds a high climbing free acid curve in the fractional test meal and a greatly exaggerated output of free hydrochloric acid over a fasting period. Psychosomatists have attempted to define a personality type—the energetic and hard-working aggressive "successful" person with retarded emotional development and a conflict aroused by a life situation which finds an outlet in gastric symptoms.

As age advances the incidence of gastric ulcer increases, and women are affected almost to the same extent as men; undernourishment is usual and a social distinction is evident, the lower social classes being more often affected. The clinical story is not nearly so characteristic and may baffle the clinician until the radiologist assists him. The clinical pathologist is no help in the diagnosis, as the fractional test meal result is either normal or subnormal. Psychosomatists are less confident in their descriptions of a personality type.

If we concede that œsophageal and jejunal ulcers occur only after surgery or when there is a defective cardiac sphincter, we can then confine our attention to the treatment of duodenal and gastric ulcerations and their complications.

Duodenal Ulceration.

The medical treatment of an established duodenal ulcer is based on the following principles:

1. Neutralization of hydrochloric acid by (*a*) food given at regular intervals, (*b*) alkaline or antacid medication, and appropriate associated measures such as intragastric drip therapy.

2. Suppression of hydrochloric acid production and reduction of peristalsis by the use of (*a*) atropine, (*b*) ganglionic blocking agents, such as the hexamethonium compounds and "Banthine", (*c*) deep irradiation.

3. Suppression of neurogenic factors by (*a*) rest, (*b*) sedation, (*c*) psychotherapy at physician level.

Despite its critics this planned regime is successful in the vast majority of cases. Most patients respond at once with rapid healing of the ulcer. Indeed, if rapid relief of symptoms is not forthcoming, the diagnosis should be reviewed or some other factor is operating as a complication.

Most physicians would agree about the bland frequent feeds, the protein and vitamin content of which should be generous. A more personal interest in the nutritional quality of the diet and the abolition of meaningless name tags to diets, such as Sippy and Meulengracht, would give better therapeutic results. A general review of all hospital ulcer diets in the light of present knowledge would be refreshing both to the patient and to the hospital finances.

Alkalis and antacids given in the usual dosage have only a transitory effect in neutralizing hydrochloric acid, but are useful therapeutic agents in the relief of pain. Intragastric drip administrations of milk and alkalis or regular night feeds may be helpful in those cases in which pain occurs at night and in which symptoms persist after ten to fourteen days. When patients who have pain occurring at night are treated in their homes, the intragastric drip apparatus can be replaced by the "Thermos" flask and alarm clock by the bedside. In this way the patient may give himself two-hourly feeds throughout the night without losing much sleep. In hospital practice one has found it less discomforting to the patient to be half woken by a nurse with a cup of milk every two hours during the night than to have an intragastric tube. Within a few days night pain is usually abolished.

Atropine is always of value, but is generally not given in sufficient quantities to be effective. A minimum of 30 and up to 50 minims of the tincture is required daily. The new ganglionic blocking agents are helpful, but do no more than atropine, and there are often unfortunate side effects which limit their usefulness. In intractable cases with very high free acid production, deep X-ray therapy directed to the abdomen to produce temporary achlorhydria has been tried successfully in selected cases, but considerable skill is needed and the procedure is not without danger.

Rest to the body and mind is of especial benefit. There are often reasons why bed rest may be impractical, and ulcers may be made to heal with the patient ambulatory and even working. Nevertheless, bed rest undoubtedly accelerates healing in most cases. Sedation is generally required, while sympathetic and frank discussion of the patient's background, domestic and social, may result in the easing of the supposed underlying conflict. Smoking is better avoided altogether.

Ryle rightly comments that "the length of time elapsing between first appearance of symptoms and diagnosis is usually to be reckoned in years rather than months". It should be stressed that the earlier treatment is instituted the better, and if effective healing is obtained early in the process the likelihood of subsequent breakdown is less.

The prevention of recurrence requires personal studies by the physician of the constitution of the patient, his habits of life and his environment, especially in the domestic sphere. Regularity and moderation in all things form the cornerstone of the healed duodenal ulcer patient's existence, as the conditions favourable for further ulceration appear to remain indefinitely. In my view these simple but rational measures are insufficiently exploited by their medical advisers and inadequately understood by the patients themselves.

In summary, then, the treatment of uncomplicated duodenal ulceration is essentially medical, and the response to rational medical treatment is good. Unhappily in the natural course of the disease there are recurrences, and with each recurrence scarring and deformity of the cap occur bringing less tendency for healing and favouring further breakdown.

When a patient is first examined and his condition diagnosed, the following factors should be considered: (i) date of commencement of symptoms; (ii) number of relapses; (iii) number of hæmorrhages, if any; (iv) evidence of penetration beyond the gastric muscle; (v) presence of an underlying anxiety state or psychosis or personality defect; (vi) previous surgery, if any.

With the possible exception of perforation, all patients suffering from duodenal ulcer will benefit by medical treatment and a period of waiting in order to assess these factors. It is remarkable how chronic penetrating ulcers will heal with prolonged rest; and even if surgery is decided upon, preliminary medical treatment will reduce the size of the ulcer and the gain in weight will improve the patient's prospects.

[1] Read at a meeting of the New South Wales Branch of the British Medical Association at Orange on October 25, 1952.

Chronic Duodenal Ulceration.

Realizing that every patient must be considered individually, in general one holds the following views regarding the treatment of chronic duodenal ulceration.

1. Except in cases of perforation, operation in the young age group should be avoided.

2. A careful estimate of the personality is essential, and the presence of personality defects and psychotic tendencies should be recognized, as surgery in these cases is usually a failure.

3. Hæmorrhage should be treated medically in most cases, but in persons over forty years of age with recurrent bleeding and especially if hypertension exists, surgery must be seriously considered and consultation between physician and surgeon take place at an early stage. If a severe hæmorrhage occurs in a patient over the age of forty years surgery is justified.

4. If obstruction exists, a short preliminary period of medical treatment should determine how much is due to functional or spastic causes and how much to organic fibrosis. Aspiration and measurement of gastric contents three hours after the evening meal give a good estimate and benefit the patient. When organic obstruction exists and is persistent, surgery is always necessary.

5. Recurring chronic duodenal ulceration without hæmorrhage or obstruction should be treated medically unless there is evidence of penetration into the head of the pancreas, when surgery becomes essential. In selected cases without penetration, surgery may be indicated for economic or other reasons, but these should be kept to a minimum.

6. The operation of choice at present is partial gastrectomy. In a few selected cases of obstruction gastroenterostomy may be all that is required. Surgeons sometimes neglect to instruct their patients on dietary rules after operation, so that doubt and confusion follow. The instructions should be clearly understood by the patient. The oft-quoted indication, "failed medical treatment", should not be used. It should be replaced by a more definitive indication, such as penetration or obstruction. The term is a bad one and its continued use unjustifiable.

Ryle summed up his views on the indications for surgery in duodenal ulceration just twenty years ago as follows:

> My present policy . . . is to advise gastro-jejunostomy in cases of pyloric stenosis; in cases of long standing duodenal ulcer in which clinical history, X-rays, and test meal proclaim gross scarring or anchorage and a tendency to slow transit of food without serious stasis, and in which there has been recurrence in spite of one or more strict courses of medical treatment; in cases presenting the syndrome of anchorage to or erosion of the pancreas; and in cases of concomitant duodenal ulcer and duodenal ileus . . . . My policy is to oppose surgical treatment in youthful cases; in non-obstructive cases with short histories and adverse pedigrees or not previously accorded a strict medical treatment; in cases with recent hæmorrhage, lacking other indications; and in cases in which X-ray and test meal show gastric hurry . . . .

If one substitutes partial gastrectomy for gastro-jejunostomy it will be seen that our present views have not altered much in the last twenty years.

It is noticeable that in the literature of the United Kingdom and the United States over the past few years there has been a general trend of opinion towards surgery in duodenal ulceration at a much earlier stage than is generally entertained in this country. Indeed, Sir Arthur Porritt makes this oblique statement in a presidential address to the Hunterian Society in 1951: "While by no means advocating that surgery should be the primary treatment of ulcer, I do suggest that present-day conditions weigh the balance in that direction"—a truly remarkable statement, which is the theme of his address.

The results of gastrectomy in duodenal ulceration must be determined on the basis of operative mortality, freedom from symptoms, return of weight to normal, ability to work successfully at pre-operative employment and freedom from relapses of ulceration. Unfortunately, the excellent results claimed by some surgeons are not substantiated by others in the terms stated. When remarkably good results are claimed one is doubtful about the selection of cases, for it is the experience of most people that the patients who do well medically will also do well surgically. One therefore feels that the increased surgical trend in uncomplicated duodenal ulceration should be viewed with considerable reserve and caution by physicians. One is also mindful of the possibility of gastrectomy being performed on the basis of radiological diagnosis alone in such circumstances. Surgical papers, even when written by eminent men, should present facts rather than dogmatic statements based on personal feeling when claims are made concerning the value of surgery in duodenal ulceration. The facts can be arrived at only by careful follow-up of such patients by the surgeon concerned and an independent physician.

Gastric Ulceration.

Gastric ulcer has certain points of difference from duodenal ulcer. It tends to appear later in life and in women almost as often as men. Undernourishment is the rule, and there is often associated pulmonary, renal or cardiovascular disease. The diagnosis is not established with the same certainty as in duodenal ulcer and may be missed for some time. The middle-aged woman with intermittent backache presents such an example. Differentiation from gastric carcinoma may be difficult or impossible. The problem is not how many simple ulcers become malignant, but how many cancers present as gastric ulcers. In our experience the size of the ulcer is not a good criterion of malignant change.

The newly diagnosed patient with gastric ulcer should be considered from the following aspects: (*a*) length of history and number of relapses; (*b*) site of ulcer; (*c*) evidence of penetration into neighbouring organs—diaphragm, pancreas, liver *et cetera*; (*d*) number of hæmorrhages, if any; (*e*) presence of associated disease; (*f*) previous surgery, if any; (*g*) possibility of malignant change.

When this information is collected, planned treatment can be decided upon. Uncomplicated gastric ulceration heals readily on medical treatment, but a later recurrence is generally the rule if the patient returns to his previous mode of living. When associated with diabetes or severe hypertension or chronic chest disease, the management will be difficult.

Treatment of an established uncomplicated gastric ulcer follows similar principles to those laid down for uncomplicated duodenal ulcer. Greater emphasis is made on a high-protein high-calorie diet with added vitamin supplements. Bed rest and food are the most important requirements. Healing may be followed gastroscopically with advantage in certain cases. Prevention of recurrences depends upon the maintenance of bodily weight by the continued intake of adequate Calories, proteins and vitamins and the abolition of smoking and alcohol.

Treatment of recurrent uncomplicated chronic gastric ulceration is probably best dealt with surgically after one or two recurrences. Surgery should not be long delayed if moderate or severe bleeding has occurred in a patient over forty years of age. Penetrating gastric ulcers will generally heal in time, but they usually recur at the old site, so that surgery is generally indicated.

In general, one has the following views concerning the place of surgery in gastric ulcer: (*a*) When any reasonable doubt exists as to the possibility of malignant change. (*b*) When extragastric penetration has occurred into the pancreas, liver *et cetera*. (*c*) If one major hæmorrhage has occurred in a patient over the age of forty years. (*d*) If definite indications of healing (radiological and gastroscopic) are absent after one month's bed rest. (*e*) If there are one or two recurrences in a patient over forty years of age. The physician feels happier in recommending surgery in these cases, as the patients are in an older age group, the ulcer recurrence rate is exceedingly small

and the danger of malignant change is removed. The operative results are usually good.

Complications.

With one exception all complications of peptic ulceration are surgical problems, so that hæmorrhage only will be briefly discussed.

Hæmorrhage.

Hæmorrhage occurs as a complication in a quarter of the cases of peptic ulcer and approximately equally in gastric and duodenal types. It is usually the presenting and only symptom of acute peptic ulceration.

In cases of hæmorrhage from peptic ulceration careful consideration should be given to the following factors: (*a*) age of the patient; (*b*) ulcer site; (*c*) associated complications, such as cardio-vascular, thoracic and renal disease; (*d*) severity of the hæmorrhage. For patients under the age of forty years the mortality is low whatever the treatment (2% to 4%) and severe bleeding is uncommon. With increasing age there is an increasing mortality, which rises to around 25% to 45% at seventy years. The mortality associated with bleeding from gastric ulcers is higher than from duodenal ulcers. Ulcers situated on the posterior wall lesser curve may erode the left gastric artery, or the splenic artery if on the mid-posterior wall towards the greater curvature. The presence of associated cardio-vascular or other diseases greatly increases the hazard of hæmorrhage.

The severity of the hæmorrhage may be judged from the vomited blood and the degree of shock as evidenced particularly by blood pressure and pulse readings. Circulatory failure should be treated without delay, and this applies particularly to old people, with whom there is added risk of cardiac failure, cardiac infarction and blindness.

The key year in improvement in therapy was 1935. In that year Kekwick and Marriott introduced drip transfusion and Meulengracht advocated liberal feeding. At the same time Gordon-Taylor began to demonstrate the value and place of surgery. The subsequent years up to the present have seen the better understanding and use of these three advances, and the over-all mortality has been reduced to between 6% and 9%.

Although there are notable exceptions and each patient must be considered individually, generally one has found the following principles to be satisfactory in cases of bleeding peptic ulcer: (*a*) Bleeding in patients under the age of forty years seldom gives cause for anxiety, and only about one-half require blood transfusion. (*b*) Bleeding in patients over the age of forty years frequently gives cause for anxiety, and almost all require blood transfusion. (*c*) Transfusion should be given when there is evidence that one litre of blood has been lost, or if the hæmoglobin value falls below eight grammes *per centum* after rehydration, or if shock is present. Transfusion at the rate of 500 millilitres eight-hourly is usually satisfactory, although more rapid rates may be necessary. (*d*) Consideration should be given to surgery in all cases, especially with patients over the age of forty years, if a second major hæmorrhage occurs in hospital, and an early consultation with the surgeon is essential.

This brief survey of opinion based on experience in many centres and on our own experience at Royal Prince Alfred Hospital is presented in general terms. The arbitrary age period of forty years is often used, but naturally there will be exceptions, and every patient will be assessed individually, regard being had to the many factors which are so important in peptic ulceration. While no effective form of prophylaxis against peptic ulceration has as yet appeared on the horizon, one may confidently predict that medical advances will lessen the burden this man-made disease is laying on man, on industry and on society generally.

References.

DOLL, R., AVERY JONES, F., and BUCKATZSCH, M. M. (1951), "Occupational Factors in the Ætiology of Gastric and Duodenal Ulcers", Medical Research Council Special Report Series, Number 276.

RYLE, JOHN A. (1936), "The National History of Duodenal Ulcer", in "The Natural History of Disease", Oxford University Press, London.

PORRITT, A. (1952), "A Gastric Gossip", *Brit. M. J.*, 2: 107.

MERRIOTT, H. L., and KEKWICK, A. (1935), "Continuous Drip Blood Transfusion with Case Records of Very Large Transfusions", *Lancet*, 1: 977.

MEULENGRACHT, E. (1935), "Treatment of Hæmatemesis and Melæna with Food", *Lancet*, 2: 1220.

GORDON-TAYLOR, G. (1937), "The Problem of the Bleeding Peptic Ulcer", *Brit. J. Surg.*, 25: 403.

SURGICAL ASPECTS OF THE TREATMENT OF PEPTIC ULCER.[1]

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As you know, medical and surgical treatment are not conflicting forms of therapy; they are complementary. Ideally, with ulcer patients there is the closest teamwork between physician and surgeon, not to mention the ward sister, dietitian, almoner and follow-up clerk. In gastro-enterological clinics at the present day only about one in six ulcer patients comes to surgery. Each must be studied and treated individually; there are no easy rules of thumb governing the selection of those for surgery.

Indications for Surgery.

Broadly speaking, surgery is confined to patients with recurrent and complicated ulcers. As for recurrent ulcer, it has been said that the physician's problem is not to heal the ulcer—very few ulcers fail to heal with intensive treatment—his problem is to keep it healed. So, it is not so much the patient's present ulcer that counts as his history. With duodenal ulcer patients, if there is a history of over three to five years, with much pain and loss of working time, surgery is often indicated. With gastric ulcer, recurrence after one full course of medical treatment is usually enough. The complications of peptic ulcer requiring surgery include perforation, obstruction, hæmorrhage, penetration and fistula formation.

If we are to get good results from surgery, we must limit its use to these complicated and recurrent ulcers. And at all costs operations such as gastrectomy must be avoided in patients with small or dubious ulcers, especially if there are prominent psychoneurotic symptoms. When the mental upset is large and the peptic ulcer is small, the results of surgery are poor.

Our guiding principle as surgeons should be to make sure that the symptoms are due to the ulcer. Persistent severe pain then usually indicates that the ulcer is penetrating. The results of surgery are excellent when carried out for these large penetrating ulcers causing severe pain.

E.W., a male, aged fifty years, occupies a responsible post in a flour mill. He complained of indigestion for eleven years and, for the past three months, of persistent severe pain in the left hypochondrium radiating through to the back. He had lost one and a half stone in the past eighteen months. While awaiting operation in hospital and receiving medical treatment, his pain was very severe and at times required morphine for its relief. X-ray examination after the swallowing of barium showed a large gastric ulcer, and penetration into the pancreas was confirmed at subtotal gastrectomy on September 25, 1951. He was discharged from hospital twelve days after operation and was doing his full work after six weeks. Since the operation he has been quite free of pain and regrets only that he did not undergo it years earlier.

Subtotal gastrectomy affords dramatic relief in patients with penetrating gastric ulcer.

Perforation.

What is the management of a perforated ulcer? Sometimes we meet this emergency in conditions inopportune for surgery, or anyway involving considerable delay. A

[1] Read at a meeting of the New South Wales Branch of the British Medical Association at Orange on October 25, 1952.

Ryle's or similar tube should be passed at once and the stomach kept empty by repeated aspiration. Thus the escape of food and gastric juices through the perforation is minimized. Peritonitis remains localized. Such intubation should always be combined with intravenous therapy and the administration of the antibiotics.

Even in a metropolis there may be several hours' delay before the perforation is sutured. So immediate intubation and aspiration are a useful routine; they are also helpful in combating the initial severe pain and shock of the perforation.

But intubation and the antibiotics are only adjuvants to, not substitutes for, surgery. Emphatically we should not, as a routine, rely on intubation and the antibiotics as our sole therapy. Except in a few hands such reliance has resulted in a distinctly raised death rate. One reason is that the diagnosis may be erroneous, the real lesion admitting of no delay in active treatment.

Only occasionally are intubation and the antibiotics applicable as a sole form of therapy; for example, if surgical facilities are just not available, and also if the patient is elderly and a bad surgical "risk", and the perforation has been present forty-eight hours or more—in this group the death rate after surgery is very high. Intubation may also suffice if the perforation is very small, causing only a slow leak, so that clinically the existence of an "acute abdomen" is even doubtful.

In the English-speaking world, the traditional treatment has been suture closure of the perforation, and in recent years the mortality rate of this simple procedure has fallen sharply owing to improved anæsthesia, the antibiotics, intubation and so on. But it is now known that the follow-up results of this simple suture are unsatisfactory (Tovee, 1951). More than half the patients soon develop severe ulcer symptoms again. Some ulcers perforate for a second time. And when it has been a gastric ulcer that has perforated, about one patient in every twelve will return with a gastric cancer though there has been no suspicion of cancer when the perforation was sutured.

G.H., a male, aged forty-four years, came under my care with a perforated duodenal ulcer in September, 1951. Perforation had occurred twelve hours before, and his general condition was not good. Nothing more than a simple suture was attempted. Elective gastrectomy was advised subsequently, but he deferred it.

He had a previous history of dyspepsia for fifteen years, beginning with another perforation which was sutured. Between the perforations there had been five episodes of bleeding with melæna. He is a director and high executive of a big department store, and after several months' sole executive responsibility, both the other directors being abroad, he is again bleeding.

Thus, though simple suture tides the patient over his emergency it does not answer the long-term problem, and for this reason surgeons are inclining towards gastrectomy performed either at the time of the actual perforation or within six months afterwards.

Continental surgeons have led in practising primary partial gastrectomy for perforations. In a recent series of 131 patients from Holland, Nuboer's (1951) mortality rate was 3·8%. This of course compares very favourably with the mortality rate of simple suture, and we must conclude that the objections to primary gastrectomy are largely theoretical at the present day. Since follow-up studies have shown that some perforated gastric ulcers are carcinomatous, it is clear that, in this group at any rate, trial of primary gastric resection is certainly justifiable.

Obstruction.

Obstruction in an hour-glass stomach or at the pylorus, due to cicatricial contracture, to œdema round an active duodenal ulcer or to erosions, indicates surgical treatment. If there has been much vomiting, alkalosis may be present and, if undetected, may greatly increase the operative risk. This alkalosis is silent clinically, but reveals itself by the presence of a high carbon dioxide combining power and blood urea content, and a low blood and urinary chloride content. It is corrected by intravenous salt therapy, controlled by readings of the carbon dioxide combining power. Also for these patients with obstruction, pre-operative intubation and gastric lavage are indicated.

Hæmorrhage.

We have to distinguish between persistent slight hæmorrhage on the one hand and massive hæmatemesis or melæna on the other. In the acute phase of massive hæmorrhage medical therapy is generally indicated. After the emergency is past, elective gastrectomy must be seriously considered, except in women with acute gastric erosions.

What is the role of surgery in the phase of massive hæmorrhage? Modern medical therapy is very satisfactory and saves over 90% of patients. Clearly, the ideal treatment would be to pick out the patients whose condition would be fatal with medical treatment and have them all operated on early.

Unfortunately, we can only guess which patients will die with persistent medical treatment. Nevertheless, experience has shown that it is risky in patients fifty to seventy years of age with a known chronic ulcer, especially a chronic gastric ulcer, and when the initial hæmorrhage from the ulcer has been profuse. Its mortality is also higher when bleeding recurs in hospital.

Severe pain, especially if there are signs of peritoneal irritation, suggests a perforation in addition to hæmorrhage and makes operation imperative. In these patients with severe pain, myocardial ischæmia must be excluded. Superadded obstructive phenomena also tilt the balance in favour of surgery. Surgery is rarely indicated in young patients with hæmatemesis, who may therefore be observed for longer periods, even though they are bleeding severely.

D. McD., a male, aged twenty-eight years, was admitted to hospital under Dr. Stuart Allen on February 10, 1950, suffering from hæmatemesis. He had been discharged from the army in 1942 because of hæmatemesis. Subsequently he suffered from indigestion and three further massive hæmorrhages occurred, the most recent being on the day of admission to hospital, when he vomited "several pints of blood". In hospital, hæmatemesis occurred at least once in each twenty-four hour period until the fourth day, when I was asked to see him. A partial gastrectomy was performed, the lesion being a chronic gastric ulcer. Twelve litres of blood had been given up to the commencement of the operation. Recovery was uneventful.

However, procrastination in older patients with recurrent hæmatemesis may be fatal. The decision for or against surgery should be made within twenty-four to forty-eight hours of admission to hospital. If an operation is to be performed, the patient is promptly taken to the operating theatre, where he is first resuscitated with a rapid drip transfusion. He is then operated on to control the source of hæmorrhage. Its location should, if possible, be identified before the surgeon proceeds to gastrectomy, and this may necessitate wide gastrotomy or duodenotomy. In desperate cases it may be possible only to open the stomach or duodenum and transfix the bleeding point.

Bleeding gastric ulcers have a worse prognosis than duodenal ulcers because they bleed more profusely and are relatively more common in older patients. In fact, with gastric ulcer all the complications are more serious than with duodenal ulcer. Thus the tendency is to make more ready use of surgery in chronic gastric ulcer. The following are reasons why gastrectomy is, in fact, becoming accepted as the treatment of choice for chronic gastric ulcer:

1. Massive hæmorrhage is much more often fatal than with duodenal ulcer.
2. Some patients with perforations return with cancers.
3. The "peptic" ulcer is, or may become, carcinomatous.
4. The follow-up results of medical therapy are more disappointing than with duodenal ulcer. Some 50% of gastric ulcers recur in their original site within five years of "curative" medical therapy.

5. The gastric acidity is normal or low. Anastomotic ulcer following gastrectomy for gastric ulcer is almost unknown.

6. Since the ulcer is in the stomach, closure of the duodenal stump is easier and stump leakage, the chief risk of gastrectomy, is less frequent.

Malignant Degeneration.

I do not know how frequently peptic ulcers undergo malignant change or how many ulcers labelled peptic have been malignant *de novo*. But it is clear that the possibility of malignancy must be considered with all gastric ulcers, and this of course applies particularly when the ulcer is in the pyloric antrum or on the greater curvature.

The history may be misleading, as in one of my cases.

This patient, a man, aged forty-four years, had a five years' history of epigastric pain half to one hour after meals. There were intermissions of several weeks, and his pain was worse at times of stress. He raises sheep down near Hay, and complained that his indigestion was always worse when he came to Sydney for the annual wool sales. He had lost about one stone in weight (from 14 stone down to 13 stone) in the past year, but he had been working hard in a hot climate. Thus his history was merely one of recurrent dyspepsia.

Radiological examination showed a pre-pyloric ulcer. In-patient medical treatment reduced its size, but it was still present after six weeks. A gastrectomy was performed and revealed a very small pre-pyloric cancer.

In fact, of course, with all pre-pyloric ulcers we must ask ourselves: "Is this ulcer malignant?", and unless complete healing can be demonstrated radiologically or gastroscopically, gastrectomy is called for.

Surgical Operations.

The following are some of the operations used in the treatment of peptic ulcer: (i) Standard ("two-thirds") gastrectomy. (ii) Vagotomy *plus* pyloroplasty or gastro-jejunostomy. (iii) Gastro-jejunostomy. (iv) For gastro-jejunal ulcer with hæmorrhage or fistula: dismantling, repair and subtotal gastrectomy, or vagotomy alone. (v) Œsophago-gastrectomy. (vi) Gastrotomy or duodenotomy and transfixation of bleeding point. You will not want me to go into the technical details of their execution, but I should like to emphasize a few points concerning subtotal gastrectomy. Firstly, in clearing the greater curvature of the stomach it is much easier and safer to work below the arterial arcade. The feeding vessels, the right and left gastro-epiploics, are finally secured below the pylorus and at the left limit of gastrectomy respectively.

Secure closure of the duodenal stump is of vital importance; stump leakage is one of the few remaining causes of fatality following gastrectomy. If the ulcer is in the stomach and the duodenum is normal anatomically, secure closure should always be achieved. Figure I shows the technique followed by the writer. A Parker-Kerr suture of number 0 chromicized gut is first inserted over a narrow-bladed crushing clamp (Figure IA). After the clamp has been removed and the suture pulled tight, the suture is returned and tied at its point of commencement (Figure IB). The individual stitches in this layer must go down to the submucosa. Next is introduced a layer of interrupted Halsted mattress sutures of number 60 linen thread (Figure IC). Finally a tag of omentum is sutured over the line of closure (Figure ID).

The same technique can usually be employed even if an ulcer is present in the duodenum. Very large active duodenal ulcers with much surrounding inflammatory œdema are difficult, but they will rarely be encountered if sufficient time has been given to pre-operative medical treatment. With a penetrating ulcer the duodenum may of course be opened. It is then generally unwise to carry the dissection to the point where a cuff is developed beyond the ulcer. It is safer to transect the duodenum at the ulcer level and to close the open duodenum by folding its anterior wall over into the ulcer crater, a series of interrupted Lembert sutures of number 60 linen thread being used and going back through the edge of the ulcer. A second layer of interrupted Halsted mattress sutures of the same material is then used to roll the stump into the head of the pancreas, the overlying peritoneum being picked up superficially. Finally, an omental patch is drawn up over the line of closure.

Turning to the gastro-jejunal anastomosis, a simple and safe method is the antecolic type of Hofmeister valve

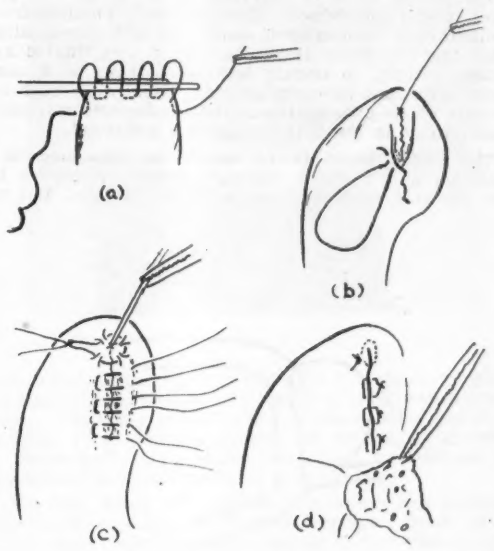

FIGURE I.
Closure of duodenal stump.

gastro-jejunostomy (Figure II). In preparing for the anastomosis, it is important to clear both curvatures of the stomach for one centimetre beyond the proposed line of resection. However, unnecessarily high ligation of the left gastric artery and stripping of the omental supports of the gastric remnant should be avoided, because if unsupported the remnant may afterwards be unduly stretched by the weight of food and the pull of the loop in the erect posture. There is evidence (Butler and Capper, 1951) that such undue stretching is partly responsible for the post-gastrectomy "dumping" syndrome, the symptoms being promptly relieved by lying down.

As for the anastomosis itself the importance of accurate suturing and of careful hæmostasis should be emphasized. In performing the gastro-jejunostomy I do not use clamps. Hæmostasis is achieved by cutting with the diathermy, ligating any vessels that bleed, and inserting an inner layer of continuous chromicized catgut interrupted at least once during its insertion. Two layers of sutures suffice, the outer layer consisting of interrupted number 60 linen thread. The segment of jejunum selected for the anastomosis lies about twelve inches beyond the duodeno-jejunal flexure, and it should come up in front of the transverse colon without any tension. I would emphasize the importance of careful attention to the mechanics of the anastomosis. The jejunal loop should lie straight and evenly against the stomach wall (Figure III). There should be no twisting or rolling of the loop after the anastomosis is completed. The inner layer of catgut sutures should go through all the coats, but an excessively high, bunched-up cuff must be avoided, especially at the corners.

Gastro-jejunostomy.

Gastro-jejunostomy is still applicable in certain patients with a scarred duodenal ulcer causing pyloric stenosis, especially if the patient is elderly and a bad surgical "risk". Also, if there is massive inflammatory œdema round the ulcer and closure of the stump is likely to be hazardous,

a safe course at operation may be to rely on a gastro-jejunostomy. If it is thought that with relief of obstruction and stasis the gastric acid level will again become high, gastro-jejunostomy can be combined with infradiaphragmatic vagotomy.

Vagotomy.

The precise scope of vagotomy in the surgery of peptic ulcer is still not defined. Further time is required for the evaluation of its long-term results. It is, however, already clear that vagotomy alone has only a very limited application, namely, to certain bad-risk patients with anastomotic ulcer and its complications. Usually for such anastomotic ulcers dismantling of the anastomosis with partial gastrectomy is the better long-term treatment.

The vagus nerve is, of course, secreto-motor to the stomach, and a really complete vagotomy results in a low flat acid curve and prompt relief of pain. But there

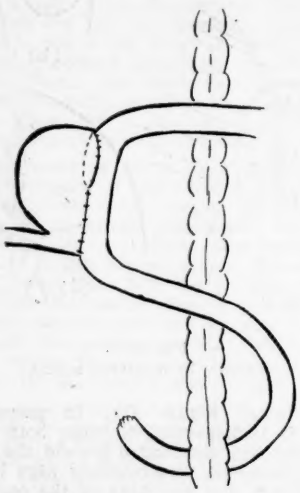

FIGURE II.
Ante-colic Hofmeister valve gastro-jejunostomy.

is also loss of motor tone, and this leads to gastric retention and stasis with much consequent discomfort. Therefore, vagotomy is now combined with some drainage operation, such as pyloroplasty or gastro-jejunostomy.

The possible application of vagotomy and pyloroplasty may be illustrated by reference to a poor-risk patient, recently under my care. He was a man, aged sixty-four years, with very severe chronic bronchitis and emphysema. He was obese and flabby, and his exercise tolerance was poor. Nevertheless, surgery was considered to be advisable because over the past nine months he had had two massive hæmorrhages from a chronic duodenal ulcer. Despite preliminary medical treatment there was much duodenal scarring around a posterior ulcer. An infradiaphragmatic vagotomy and pyloroplasty were carried out. With help from the anæsthetist and physiotherapist his chest condition stood up to the operation and his immediate post-operative result has been satisfactory; he has lost his pain and there is no acid response to an insulin test meal.

Œsophago-gastrectomy.

I should like particularly to say a word about peptic œsophagitis. The reflux of peptic juice into the œsophagus is now known to be the cause of heartburn and the regurgitation of sour acid material into the mouth. Heartburn is a common symptom. There is burning pain high in the epigastrium or beneath the sternum, which may radiate through to the back or spread up to the suprasternal notch. The patient is often obese and beyond middle age. The œsophageal regurgitation results from defective muscle control of the cardiac orifice. The pain and heartburn are worse if the patient bends over or lies down and especially if he lies on the right side. So it is felt chiefly when he is in bed at night and it may wake him from sleep.

The reflux of gastric juice produces œsophagitis, and this leads in turn to peptic ulceration. Finally, there are scarring and stenosis of the lower part of the œsophagus. Sometimes obstruction becomes extreme and requires œsophago-gastrectomy for its relief.

A man, aged fifty-eight years, was recently referred to the Unit of Clinical Investigation at the Royal North Shore Hospital of Sydney with the complaint of heartburn for five years, worse when lying in bed at night, especially on the right side. More recently he had developed difficulty in swallowing, regurgitation and severe pain beneath the lower part of the sternum. In the three months prior to admission

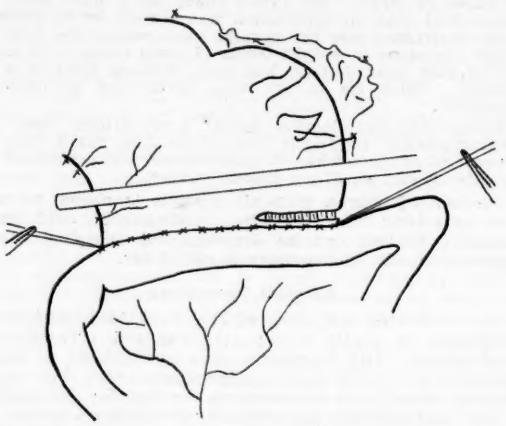

FIGURE III.
Approximation of stomach and jejunal loop by interrupted linen sutures (posterior layer).

to hospital he had lost over a stone in weight. His pre-operative X-ray examination showed considerable narrowing of the lower part of the œsophagus. An œsophago-gastrectomy was performed, the œsophago-gastric anastomosis lying at the level of the sixth dorsal vertebra. He has had complete relief from his pain and dysphagia, but screening examination shows that regurgitation still occurs when he lies on his right side. With the head of his bed raised eight inches on blocks he sleeps quite comfortably, and he has been advised to use such blocks permanently.

I mention peptic ulceration of the œsophagus not because its treatment is commonly surgical, but rather because, with œsophagitis, it is an important source of dyspepsia and frequently passes unrecognized. It will not be detected unless a careful history is taken. Œsophageal regurgitation will also be missed in a barium meal examination unless special steps are taken by the radiologist, as detailed by Johnstone (1952).

Results of Standard Gastrectomy in Peptic Ulcer.

In experienced hands the mortality rate of gastrectomy for ulcer is now very low, being less than 2% in organized clinics. Thus elective gastrectomy carries a clearly lower mortality rate than that of massive hæmorrhage or perforation.

As already indicated, good results depend on the careful selection and preparation of patients for surgery. The most grateful patients are those who have suffered from severe pain for years and in whom a large penetrating gastric or duodenal ulcer is demonstrable; some will have begged for an operation to be done. About nine out of ten of such patients lose their pain after gastrectomy and are delighted with the result. Their gratitude may be almost embarrassing.

After gastrectomy, about three out of four patients can return to their former occupations untroubled by repeated loss of earning power. About two out of three gain weight, but typically the post-gastrectomy subject is not obese, and failure to gain weight may be a minor cause of dissatisfaction. Standard gastrectomy involves a disability time of two to five months, but recently I had a patient whose total loss of working time was only five weeks. Most patients can eat anything after the operation; in fact each patient should be told and encouraged to eat a normal diet even before leaving hospital. Of course, overloading of the stomach remnant must be avoided. Iron and vitamins are added to the diet permanently. The patient's hæmoglobin value should be checked before he is discharged from hospital and, if low, restored fully to normal.

In the months after gastrectomy there may be attacks of epigastric tightness, weakness, palpitations, sweating and flushing soon after or some hours after meals.

Two postprandial syndromes following gastrectomy have been differentiated by Aldersberg and Hammerschlag (1947), the early and the late, the former occurring immediately after meals, the latter one to three hours afterwards. The mechanism of the first is a combination of distension of jejunal loops and stretching downwards of the stomach remnant. Thus there are epigastric fullness, nausea, palpitations, sweating and rise of blood pressure. The late post-prandial syndrome derives from hypoglycæmia and is characterized by a sensation of emptiness, hunger, tremor, exhaustion and faintness. The first is relieved by lying down and by taking smaller meals at shorter intervals; the second is relieved by food. The symptoms of the latter tend to abate spontaneously with the passage of time, but the former may be intractable.

Further, even when patients are carefully selected and screened for neuroses, some psychological cripples are found at follow-up, but these should not comprise more than 5% of patients. These problem patients complain of flatulence, pain, nausea and attacks of vomiting; screening examination fails to reveal any mechanical difficulty at the stoma. There are also general listlessness and fatiguability. But the minority of unsatisfactory results should not be allowed to obscure the excellent over-all picture. Some 90% of patients are delighted with the result, and a high proportion of these are able to forget that they have ever had a peptic ulcer.

References.

ADLERSBERG, D., and HAMMERSCHLAG, E. (1949), "Mechanism of the Post-Gastrectomy Syndrome", *J.A.M.A.*, 139: 429.

BUTLER, J. J., and CAPPER, W. M. (1951), "Experimental Study of 79 Cases showing Early Post-Gastrectomy Syndrome", *Brit. M. J.*, 1: 1177.

JOHNSTONE, A. S. (1952), "Radiology of the Œsophagus", in "Modern Trends in Gastroenterology", Butterworth's, London.

NUBOER, J. F. (1951), "Primary Partial Gastrectomy for Perforated Ulcer", *Lancet*, 2: 952.

TOVEE, E. B. (1951), "Late Results of Surgery in Perforated Duodenal Ulcer", *Arch. Surg.*, 63: 408.

AN ENDOTRACHEAL HEAD HARNESS.

By RONALD V. PRATT,

Perth.

DESIGNED originally (and still of principal value in this field) to facilitate tonsillectomies when the patient had been intubated through the mouth, the appliance to be described has now been in use for more than two years and has proved to be, in simplicity and efficiency, additionally useful as a means of securing both transoral and transnasal endotracheal tubes. It is especially helpful in maintaining general anæsthesia for dental surgeons and oto-rhino-laryngologists.

In use, this light stainless steel device, prior to the induction of anæsthesia, is fastened to the patient's forehead by means of an attached plastic belt. The nipple of a standard Australian endotracheal connexion is screwed back into its adapter through a hole three-eighths of an inch in diameter in the device; this provides, after induction, a rigid attachment for the anæsthetic delivery tube (such as exhale valve and corrugated hosing from a "Trilene" or ether vaporizer), which is far superior to strapping or other makeshift measures. A suitable endotracheal tube having

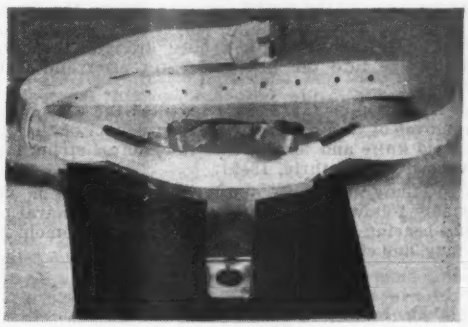

FIGURE I.

been passed, connexion is made with the nipple by means of a Magill's angle-piece and a length of thick-walled rubber tubing. The latter tubing is then slipped under one of the retaining clips, and the surgeon is invited to introduce his Boyle-Davis gag (taking care that he includes the endotracheal tube inside the curve of the gag).

With this set-up the surgeon has unimpeded exposure of one tonsillar fossa. To work on the other fossa, he has only to slide the thick tubing across to the opposite clip, when the Magill angle-piece will take up a corresponding position at the opposite angle of the mouth.

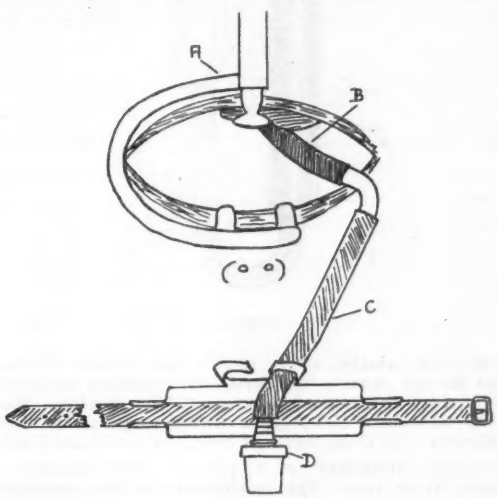

FIGURE II.

Head harness in position. A, Boyle-Davis gag; B, endotracheal tube; C, pressure tubing; D, standard endotracheal adapter.

Although primarily designed to facilitate tonsillectomy for the surgeon who insists on an orally intubated patient, this device also provides an efficient and unobtrusive holder for endotracheal tubes introduced nasally. In such cases, of course, a Magill nasal connexion must be used together with an appropriately shortened length of thick-walled tubing.[1]

[1] The head harness described may be obtained from Messrs. Felton, Grimwade and Bickford, of Perth, or from their representatives elsewhere.

SURGICAL DIATHERMY IN THE TREATMENT OF NASAL POLYPOSIS.

By ERIC GUTTERIDGE,
Melbourne.

HIPPOCRATES, in the fourth century before the Christian era, described the soft nasal polypi and the method of removal with the aid of a sponge dragged through the nasal passage into the pharynx (Stevenson and Guthrie, (1949).

Gabriel Fallopius (1523-1562) invented the wire snare for the removal of nasal polypi. The physicians of the Middle Ages used knife and forceps and the knotted string method (Stevenson and Guthrie, 1949).

Polypectomy by snare and grasping forceps is almost invariably followed by recurrence. Radical removal of the polypus-bearing sinus mucosa by antral and ethmoid operation may not effect a cure.

FIGURE I.

Chemical caustics, silver nitrate and chromic acid solutions do not cause a retrogression. Radium applications may reduce the rate of progress by the resulting fibrosis of the portion of the polypus within range of the radium applicator. All these forms of treatment are unsatisfactory.

Surgical diathermy of a polypus will coagulate and destroy it *in situ*. The employment of the conventional diathermy with the indifferent electrode on the back and the fulgurating electrode in the polypus will be effective in the destruction of the polypus, but the current in its path to the distal electrode may affect contiguous structures, the lateral nasal wall, the septum, the eye or the optic nerve. However, if the terminals are connected each to a needle, so placed in a non-conducting rod as to be four millimetres apart, the spark will be across the separated points and the heating effects will be localized to this arc and will not affect the adjacent structures.

The biterminal electrode of Hurd and Morrison fulfils this requirement. It is employed to coagulate and fibrose the inferior turbinal in hypertrophic rhinitis of the turbinal. The needles are inserted into the inferior turbinal, the current is passed and a burn of the track of the needles ensues with resultant fibrosis and contraction of the turbinal.

The biterminal electrode is employed in the treatment of polypi. Microscopic sections made of polypi treated by diathermy have demonstrated that the heating and coagulation effects were limited to the area between the points of the terminal needles. There was no spread of coagulation.

Polypi in the ethmoid cells have been treated by diathermy without deleterious effects. The procedure may be regarded as without hazard, provided that a minimal current is not exceeded.

The conventional diathermy machine or an electro-surgical unit is employed. The current is regulated for "mild coagulation". A minimal sparking across the points is requisite.

To anæsthetize the nasal passage, the nose is sprayed with a local anæsthetic, such as "Decicain" (2%) with adrenaline (1 in 1000), three parts to one. Cocaine and adrenaline paste or 10% applications are made to the anterior ethmoidal nerve in the roof of the nose and to the

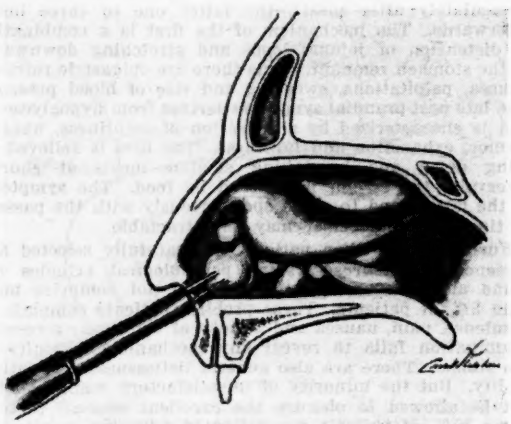

FIGURE II.

spheno-palatine ganglion, if it is not obstructed by the polypoid mass. The nasal septum receives an application. The insensitivity of the polypi makes the procedure relatively painless.

The needles of the biterminal electrode are pressed into the polypus presenting. The current is switched on. The polypus is seen to blanch between and around the needles; it becomes turgid and rigid. Coagulation progresses to desiccation. The process should not be continued to dryness, for sparking will disturb the patient.

The coagulated portion of the polypus is grasped and removed with alligator forceps. Further diathermy and removal of the polypus continue until the middle meatus is cleared. A light fulguration of the base of the polypus will induce fibrosis and diminish regrowth of the polypus. The treatment may be repeated on the remaining polypi after a fortnight.

The polypi may be treated by diathermy without removal. Quiet necrosis follows the desiccation; the treated portion separates and is discharged as a crust. The polypus shrinks from the induced fibrosis. Subsequent diathermy of the remaining polypi follows. Little anæsthetization is required.

The diathermic current seals the blood vessels of the polypi. There is no hæmorrhage at the time of treatment or in the reactionary or secondary periods. There is therefore no necessity to insert any nasal packing.

Resultant inflammatory reaction is minimal, a mild congestion of the nasal mucous membrane passing off in a few days. There is no pain. A simple menthol and paraffin oil instillation is given.

Biterminal surgical diathermy affords a method of rapid, painless and bloodless removal of the soft nasal polypus.

Reference.

STEVENSON, R. SCOT, and GUTHRIE, D. (1949), "A History of Oto-Laryngology".

BLOOD COUNTS OF ELDERLY SUBJECTS.

By MARY K. LAING,

Red Cross Blood Transfusion Service, Sydney.

IT has been found that there is a progressive decrease in the mean hæmoglobin values of both sexes with increasing age (Walsh *et alii*, 1951). The decrease first became apparent in males aged between sixty and sixty-nine years and in females aged between seventy and seventy-nine years. The mean values of subjects of both sexes aged between eighty and eighty-nine years was 92% of the mean values of all subjects. Full hæmatological investigations were performed on a group of elderly people to determine whether the fall in the hæmoglobin value was associated with a change in the red corpuscular and hæmoglobin contents.

Subjects and Methods.

Volunteers were chosen from inmates of the Lidcombe and Newington State hospitals and of a private nursing home. Subjects suffering from diseases not characteristic of old age were excluded, and 30 males and 30 females were tested. From each subject five millilitres of venous blood were obtained from the cubital vein and placed in tubes containing crystals of sodium and potassium oxalate as described by Wintrobe. An interval of six hours usually elapsed between collection and the performance of the tests. The hæmoglobin values were determined on a photo-electric colorimeter described by Walsh *et alii* (1951), and duplicate readings were obtained from each sample. Four red cell counts were performed on each sample, two dilutions of the blood being used in each instance. Hæmatocrit determinations were performed in Wintrobe tubes, which were centrifuged for forty minutes at 3500 revolutions per minute.

Results.

The results of all tests are shown in Table I. It will be seen that the mean figures for the red cell count, hæmoglobin concentration and hæmatocrit value are below the mean values found in subjects aged less than sixty years. The calculated indices and the white cell counts are within normal range. The standard deviations of all tests are similarly higher than those found in the general calculations.

In Table II an analysis is presented of the figures for 12 males with hæmoglobin values less than 13 grammes *per centum* and for nine females whose hæmoglobin values were less than 11·7 grammes *per centum*. The average age of the males was 78 years and of the females 76·5 years. It will be seen that the mean corpuscular volume and the mean corpuscular hæmoglobin concentration are in both sexes within the normal range as given by Wintrobe (1947).

Discussion.

The anæmia seen in these elderly subjects is mild in degree, normocytic and normochromic in nature. These findings would seem to indicate that there is no gross iron deficiency and no deficiency of liver principle. It would hardly be expected, therefore, that therapy with iron or liver extract would increase the hæmoglobin and red cell counts. It is possible that the process of aging has produced a retardation of hæmolytic function, the marrow tissue being affected in the same way as are other tissues. On the other hand, a reduced hæmoglobin and red cell synthesis may be the result of diminished activity that lessens demands for oxygen transportation in old age. A third but unlikely possibility to account for the mild

TABLE I.

Observation.	30 Males.			30 Females.		
	Mean.	Standard Deviation.	Standard Error, Mean.	Mean.	Standard Deviation.	Standard Error, Mean.
Red blood cell count	4,750,000 per cubic millimetre.	0·59	±0·11	4,460,000 per cubic millimetre.	1·41	±0·26
Hæmoglobin value	13·4 grammes per 100 millilitres.	1·62	±0·29	12·726 grammes per 100 millilitres.	1·62	±0·29
Hæmatocrit	40·33	5·21	±0·96	38·13	3·96	±0·72
Mean corpuscular volume	84·93 cubic μ	2·95	±0·54	85·51 cubic μ	3·26	±0·49
Mean corpuscular hæmoglobin	28·17 γγ	4·11	±0·75	28·3 γγ	1·35	±0·25
Mean corpuscular hæmoglobin concentration	33·07%	1·56	±0·28	33·2%	1·19	±0·22
Total white blood cell count	7550 per cubic millimetre.	1315	±234·6	8366 per cubic millimetre.	1836	±335·2
Neutrophile cells	4631 per cubic millimetre.	947	±172·9	5248 per cubic millimetre.	3645	±665·5
Lymphocytes	2235 per cubic millimetre.	399	±72·85	2316 per cubic millimetre.	578	±105·5
Colour Index	0·97	0·05	±0·009	0·99	0·20	±0·37

TABLE II.

Observation.	12 Males, Hæmoglobin Value 13 grammes *per Centum* and Under.			Nine Females, Hæmoglobin Value 11·6 Grammes *per Centum* and Under.		
	Mean.	Standard Deviation.	Standard Error, Mean.	Mean.	Standard Deviation.	Standard Error, Mean.
Red blood cell count	4,280,000 per cubic millimetre.	0·49	±0·14	3,940,000 per cubic millimetre.	0·28	±0·093
Hæmoglobin value	11·72 grammes per 100 millilitres.	1·49	±0·43	10·83 grammes per 100 millilitres.	0·87	±0·29
Hæmatocrit	35·96.	4·39	±1·27	33·2.	2·26	±0·75
Mean corpuscular volume	83·93 cubic μ.	2·92	±0·84	84·89 cubic μ.	3·41	±1·14
Mean corpuscular hæmoglobin	27·33 γγ.	1·26	±0·36	27·46 γγ.	1·14	±0·38
Mean corpuscular hæmoglobin concentration	32·29%.	1·02	±0·55	32·69%.	1·56	±0·52
Colour index	0·94.	0·049	±0·014	0·96.	0·07	±0·023

anæmia is the presence of chronic infection. This, however, does not seem to be more common in elderly subjects than in subjects in the younger age groups. Determination of the serum iron values and of the erythrocyte protoporphyrin values might be useful in further investigations.

Summary.

A reduction in the red cell counts, hæmoglobin and hæmatological values of subjects aged over sixty-five years has been found in both sexes. The values for the mean corpuscular volume and the mean corpuscular hæmoglobin concentration were, however, within normal limits. The slight anæmia seen in these subjects is normocytic and hypochromic.

Acknowledgements.

The writer wishes to acknowledge the facilities of the New South Wales Red Cross Blood Transfusion Service and the interest and cooperation of Dr. L. Sharfstein, of the Newington State Hospital, and Dr. G. S. Pro-Copis, of the Lidcombe State Hospital.

THE USE OF CONTROLLED HYPOTENSION COMBINED WITH THE LOCAL USE OF ADRENALINE AS AN AID TO THE FENESTRATION OPERATION.

By Denis O'Brien, F.R.C.S. (Edinburgh), F.R.A.C.S., L.O., and William H. J. Cole, D.A., *Melbourne.*

The fenestration operation is one which is greatly facilitated if bleeding is minimal, and various anæsthetic techniques with this aim have been described (Hutchinson, 1950, and correspondence following). With the advent of drug-induced hypotension this method was applied to the fenestration operation (Hughes, 1951).

The method to be described is believed to be novel, in that a modest lowering of blood pressure is reinforced by the local use of adrenaline, a combination which in our hands has been very effective. The infiltration of the soft tissues with adrenaline, either in saline or in "Novocain" solution, is employed by most surgeons as an adjunct to whatever anæsthetic technique is used for this operation. This step was at first omitted when it was decided to use hexamethonium iodide (C6), because on first sight one would suppose that the injection of adrenaline would defeat the purpose of the procedure by antagonizing the hypotensive drug and raising the blood pressure (Bromage, 1952). However, it was found that the bleeding from the soft tissues could still be troublesome, so it was decided to revert to the former practice of using the local injection. Careful blood pressure recordings taken in such cases suggest that when adrenaline is infiltrated into the tissues in dilute solution it does not enter the circulation at a rate sufficient to produce measurable change in blood pressure. (One of us, W.H.C., in conjunction with Dr. I. McConchie, thoracic surgeon, has applied this method to thoracic surgery, in which cases amounts of the order of 120 millilitres of 1 in 300,000 adrenaline solution are infiltrated into the tissues. Even in this quantity the adrenaline has not been found to counteract the hppotensive drug.)

Details of Method.

The patient is examined pre-operatively to determine physical fitness, especially cardio-vascular. The anæsthetic itself is conventional: induction is with thiopentone and "Flaxedil", followed by an endotracheal nitrous oxide and oxygen anæsthetic supplemented with pethidine given intravenously. When the anæsthesia has become stabilized after induction, the systolic blood pressure is taken by the oscillation method, a sphygmomanometer with a clock-faced gauge being used. Twenty milligrammes of C6 are injected intravenously, and in three minutes the blood pressure is measured. If necessary, further 10-milligramme injections of C6 are given. A reduction of systolic blood pressure to about 100 millimetres of mercury is desired, and this can be achieved by small doses. (This is in contradistinction to drastic falls to a systolic blood pressure of 50 to 60 millimetres of mercury, which are frequently difficult to attain even with large doses of C6 and extremes of posturing.) The patient remains horizontal. The soft tissues of the operation site are now infiltrated with a 1 in 2000 amethocaine solution and 1 in 100,000 adrenaline hydrochloride solution, and the operation is commenced. Frequent blood pressure recordings are taken, and if a tendency for the blood pressure to rise is accompanied by significant bleeding, further injections of 10-milligramme doses of C6 are made. Additional amethocaine and adrenaline solution may be injected, or during the bone work adrenaline gauze may be applied to the bleeding points.

It is believed that such small reductions in blood pressure are not accompanied by any added risk to the patient. The post-operative general condition of the patients who have been treated in this way has been very good.

Summary.

A method of minimizing hæmorrhage in fenestration operations is described, in which a slight degree of hypotension is reinforced by the use of adrenaline injected and applied at the operation site.

References.

Bromage, P. R. (1952), "Comparison of Vasoactive Drugs in Man", *Brit. M. J.*, 2: 72.

Hughes, G. (1951), "Use of Pentamethonium and Posture to Reduce Bleeding in Fenestration Operations", *Lancet*, 1: 666.

Hutchinson, B. (1950), "Anæsthesia for Fenestration Operation", *Brit. M. J.*, 1: 436.

Reports of Cases.

A FATAL CASE OF STILL'S DISEASE ASSOCIATED WITH WATERHOUSE-FRIDERICHSEN SYNDROME DUE TO PNEUMOCOCCAL SEPTICÆMIA.

By L. J. A. Parr, Eva A. Shipton and Esme H. Holland, *Sydney.*

The study of the relationship of the suprarenal glands to the other endocrine glands has received such an impetus from the work of Selye (1950) that any factor, however small, which may forge another link in the chain of knowledge is of value.

The occurrence of two fatal cases of the Waterhouse-Friderichsen syndrome in two patients suffering from arthritis—one from Still's disease and the other from typical vasospastic rheumatoid arthritis—warrants a review of the literature as to any previous recorded association of these two conditions.

That this association is not common is our experience with many thousands of patients suffering from rheumatoid arthritis observed over a period of twenty years.

In the case of Still's disease here reported in detail, the enlargement of the thymus, not diagnosed radiologically before death, was verified *post mortem*, and the smallness of the spleen is an interesting observation. In the second case the report is incomplete, and the diagnosis was made clinically.

Case I.

A female child, aged sixteen years, was admitted to hospital on December 11, 1949, complaining of pain and stiffness in the lower part of the back and of stiffness in the upper and lower limbs. In a period of six months the disease, which had begun in the metacarpo-phalangeal joints, spread to the fingers, shoulders, neck, knees and feet. The back stiffness was so pronounced that she could not bend to within 18 inches of the ground, and this feature

of her condition prompted the thought of the presence of ankylosing spondylitis. The cervical part of the spine was also affected. The disease had spread rapidly. Some enlarged lymph glands were present in the cervical area, but the spleen was not palpable. A soft presystolic murmur was present and there was some evidence of myocarditis. The apex of the heart was four inches from the mid-line. Nasal examination showed the presence of atrophic rhinitis, the pharynx being dry and covered with a mucopurulent discharge. We regarded this nasal abnormality as a rarity in so young a child. Her temperature on her admission to hospital was 100° F., and the pulse rate was 120 per minute. Attempted blood culture was unsuccessful, a red cell count showed a slight degree of secondary anæmia, and her leucocyte count was 9600 per cubic millimetre (lymphocytes 2304 and neutrophile cells 6528), whilst the erythrocyte sedimentation rate was 13 millimetres (Westergren) at sixty minutes.

Therapy consisted of the administration of adrenaline in oil (0·5 millilitre twice daily) and of "Eschatin" (suprarenal cortical extract—Parke, Davis and Company, two millilitres daily).

On January 24, 1950, an adrenaline test was carried out and gave the information shown in Table I. Her erythrocyte sedimentation rate was now normal, in spite of the fact that she still had a raised evening temperature (99·2° F.). X-ray examination of the chest revealed no evidence of pulmonary tuberculosis, but the heart was slightly enlarged.

TABLE I.

Time of Test.	Total Leucocytes.[1]	Total Neutrophile Cells.[1]	Total Lymphocytes.[1]	Total Eosinophile Cells.[1]
Before adrenaline	13,400	7772	4536	402
After adrenaline—				
1 hour	17,600	6160	9504	880
2 hours ..	16,200	5832	9720	324
3 hours ..	14,600	6132	7300	438
4 hours ..	15,200	9628	4684	—

[1] All per cubic millimetre.

On February 21 the erythrocyte sedimentation rate had risen to 71 millimetres at sixty minutes (Westergren) and she was given benzyl sulphanilamide, 0·5 gramme four times a day.

On March 3 the erythrocyte sedimentation rate had fallen to 39 millimetres in the first hour, but her temperature had risen to 104° F., and the rise was accompanied by a sulphanilamide rash. Her leucocyte count was now 14,000 per cubic millimetre, and the lymphocytes numbered 3360 per cubic millimetre, whilst the blood was sterile.

On March 28 the erythrocyte sedimentation rate was eight millimetres in the first hour (normal) and her temperature had almost subsided to normal. Penicillin was next given, but owing to an attack of giant urticaria and fever it was also suspended. This child had thus shown a pronounced degree of sensitivity to both benzyl sulphanilamide and penicillin, reacting with a severe febrile reaction and rash to the former, and with giant urticaria and fever to the latter.

On April 11 her erythrocyte sedimentation rate was 32 millimetres in one hour (Westergren) and treatment was begun with small doses of gold (0·01 milligramme of "Myocrisin"). Her leucocytes numbered 9400 per cubic millimetre, whilst the lymphocytes numbered 3384 per cubic millimetre.

On May 22 the erythrocyte sedimentation rate was 45 millimetres (Westergren) at sixty minutes, her white cell count was 19,000 per cubic millimetre, and the lymphocytes numbered 5510 per cubic millimetre.

By June 13 the erythrocyte sedimentation rate had fallen to 15 millimetres (Westergren) at sixty minutes, the white cell count had increased to 20,000 per cubic millimetre, and the lymphocytes numbered 7400 per cubic millimetre, whilst the eosinophile cell count had risen to 1400 per cubic millimetre. Her myocardial condition, which had caused some concern during the febrile stage, had greatly improved under "Digoxin" therapy, and the patient left hospital on June 29. She had greatly benefited by chrysotherapy as regards her joints and general condition, and she was maintained on small doses of gold (0·05 gramme of "Myocrisin" once a week) and 500 milligrammes of ascorbic acid once a week. Radiological examination of the chest had revealed no evidence of thymic enlargement. The sacro-iliac joints were normal on radiological examination. Table II details the results of the various tests. The rise in the leucocyte count and lymphocyte count occurred concurrently with pronounced clinical improvement, and she was discharged from hospital and placed under the care of her own local doctor. All peripheral joint disability had been greatly relieved and back flexion approximated normal.

The reports on the electrocardiograms were as follows: December 15, 1949, moderate sinus tachycardia, diffuse myocardial disease; February 3, 1950, increased myocardial damage; March 23, 1950, still evidence of further myocardial deterioration; May 2, 1950, great improvement and very little evidence of any myocardial degeneration.

TABLE II.

Date.	Erythrocyte Sedimentation Rate (60 Minutes, Westergren).	Total Leucocytes per Cubic Millimetre.	Total Lymphocytes per Cubic Millimetre.
12/12/49	13	9600	2304
30/12/49	13	—	—
24/ 1/50	8	13,400	4536
21/ 2/50	71	—	—
24/ 2/50	75	12,000	2240
3/ 3/50	39	14,000	3360
13/ 3/50	35	—	—
28/ 3/50	8	—	—
11/ 4/50	32	9400	3384
24/ 4/50	51	13,400	3480
22/ 5/50	45	19,000	5510
13/ 6/50	15	20,000	7410

This period, from December 15, 1949, to May 2, 1950, corresponded with the active stage of the disease, which had been accompanied by bouts of fever and allergic manifestations. An X-ray examination of the heart at the height of the first severe febrile attack had revealed slight enlargement. After leaving hospital the child had progressed favourably with regard to her cardiac condition.

On September 16, 1950, the patient was readmitted to hospital, desperately ill, and the following history was obtained from her mother. On the previous night, at six o'clock she had refused her dinner, at ten o'clock she had had a rigor, and at five o'clock on the following morning her mother noticed that her lips and fingers were cyanosed. The family doctor visited her at 9 a.m. and noted that the cyanosis was extending and that there were extensive purpuric patches on her face and extremities. He diagnosed circulatory failure as the cause of her illness. Her blood pressure on her admission to hospital at 10 a.m. was 60 millimetres of mercury, systolic, and 40 millimetres, diastolic, and she was cold and clammy, with a rapid pulse and normal temperature. Her face was livid and covered with purpuric spots, and her lips and ears were deeply cyanosed. The hands, forearms, feet, and lower part of the legs showed cadaveric cyanosis, and there were numerous petechiæ and subcutaneous hæmorrhages over the limbs and other parts of the body. She was dyspnœic and there were râles at both lung bases. Her heart sounds were weak and the pulse rate was very rapid; the pulse itself was soft and thready. She was diagnosed as suffering from suprarenal hæmorrhage, with associated circulatory collapse, the cause of which at the time was not clear. She died less than six hours after her admission to hospital, before blood for culture was obtained. However, blood slides were prepared just before death and showed the

presence of encapsulated Gram-positive cocci in the neutrophile cells and also lying between the cells.

A post-mortem examination was performed approximately eighteen hours after death.

The lungs were congested with petechial hæmorrhages. There was no free fluid in the pleural cavities. Microscopic examination of the lungs revealed some infiltration with neutrophile cells and some Gram-positive cocci.

The spleen weighed 35 grammes; it was small and fibrous. Microscopic examination of sections revealed numerous thickened trabeculæ and a thick capsule. The usual structure was distorted from hæmorrhage into the pulp. One siderotic nodule was found.

The thymus was enlarged, the weight being 45 grammes. A few petechial hæmorrhages were present on the surface, but microscopically the structure was normal.

The suprarenals were soft and very hæmorrhagic; a few yellow areas were seen. Microscopic examination of sections revealed gross disturbance of the normal structure in both the cortex and the medulla. Hæmorrhages were present in the cortex and under the capsule, and a few Gram-positive cocci were found in some areas.

The ovaries were larger than normal and both contained large cystic spaces filled with blood. Microscopic examination of sections revealed ruptured luteal cysts filled with blood and with Gram-positive cocci in pairs and short chains amongst the red corpuscles.

Blood taken from the heart, which had not clotted eighteen hours after death, yielded cultures of Gram-positive cocci in pairs and short chains which morphologically and culturally were pneumococci.

The post-mortem findings and blood culture therefore showed that the fatal termination was due to pneumococcal septicæmia with associated suprarenal cortical hæmorrhage, the latter accounting for the circulatory collapse.

The severe allergic reaction of this child to benzyl sulphanilamide and penicillin, and the fact that she had a high lymphocyte count in the peripheral blood, with cervical glandular enlargement, should have warned us of the possibility of susceptibility to intercurrent infection.

In the light of some recent clinical experience, X-ray radiation of the thymus and mediastinal lymph glands may have been beneficial in reducing susceptibility to infection. The enlargement of the thymus, together with these findings, adds weight to a theory postulated by us that in some cases of rheumatoid arthritis, especially Still's disease, we may be dealing with a mild *status thymicolymphaticus*. It is to be noted that in this case Thorn's adrenaline test gave a positive result, in that the eosinophile cell level fell 50%, but only at the fourth hour, whilst the lymphocyte count, initially high, rose still further.

We may well ask ourselves the following questions: Was this child affected by a pneumococcus of specially virulent character, with specific toxic properties against vascular tissues and the suprarenal cortex? Or was it an ordinary pneumococcus whose ability to produce an overwhelming infection was due to lack of resistance by the host? In other words, was it the bacterium or the soil?

Case II.

A similar fulminating case occurred in a young woman, aged twenty years, suffering from a vasospastic variety of rheumatoid arthritis and examined by us some years ago. She was admitted to hospital at about 6 p.m. with a history of sudden onset with a rigor eight hours previously, followed by the appearance of a petechial rash and large purpuric patches over the body and hands. She was almost unconscious on her admission to hospital, and circulatory collapse followed, with fatal termination, in thirty-six hours. Her blood was sterile, and examination of blood films revealed neutrophile leucocytosis with numerous platelets, but no organisms. There was no evidence of hæmolytic anæmia. No post-mortem examination was obtained.

This young woman rapidly became unconscious and circulatory collapse followed. The exact cause of the suprarenal hæmorrhage was unknown.

Comment.

In both cases reported a fatal termination was preceded by typical manifestations of the disease, but in the former the bacterial cause — namely, the pneumococcus — was isolated. In the latter case no causative organism was discovered.

Discussion and Review of the Literature.

The Waterhouse-Friderichsen syndrome is characterized by its sudden onset and fulminating purpura, and by post-mortem hæmorrhages into the adrenals and other tissues.

Waterhouse in 1911 described a typical case in a child, aged eight months, from whom no organism was isolated, and in 1918 Friderichsen collected the literature to that date and described cases of his own. Glanzman (1933) called the condition "*Syndrom von Waterhouse-Friderichsen*", but it had been recognized and described accurately by numerous authors long before this time, notably by Graham Little (1901), who fully recognized the syndrome and its severity, and found streptococci in the skin from two of his subjects.

Most authorities state that the majority of the cases are due to the meningococcus, and Martland (1944) believes that the Waterhouse-Friderichsen syndrome — cyanosis, purpura petechiæ and bilateral massive hæmorrhages into the suprarenal glands—is always due to fulminating fatal meningococcæmia.

However, in not every case are there massive hæmorrhages into the suprarenal glands, and Williams (1942) has shown that there is no essential clinical difference between patients with meningococcal septicæmia and adrenal apoplexy, and the subjects with meningococcal septicæmia alone, and many instances of the syndrome have been reported with other infections, with morphological changes in the adrenals, but without massive hæmorrhage.

Karsner (1942) stated that the condition was apparently due to fulminant bacterial infections; hæmolytic streptococci, pneumococci, influenza bacilli and meningococci have been recovered in different cases.

Most authorities are agreed that, in the majority, the syndrome is of meningococcal origin.

König (1922) obtained streptococci on blood culture from a child with the syndrome.

Scheidegger (1933) recovered the streptococcus from the suprarenals, which showed hæmorrhagic infarction caused by bacterial emboli, in a boy who had a history of a slight head injury and some meningitic symptoms and purpura.

Snelling and Erb (1935) obtained growths of the *Streptococcus hæmolyticus* from some of their patients.

Anlayan, Klippel and Curtis (1950) isolated an anaerobic streptococcus from the blood of their patient.

Dudgeon (1904) found a *Staphylococcus aureus* in one case, and Weil and Azoulay (1924) reported the case of an adult male with staphylococcal septicæmia arising from a prostatic abscess, in which purpura resembling the meningococcal type was present.

In Sachs's second case (1937) the post-mortem blood culture yielded a pneumococcus, type I, while Nobecourt and Mathieu (1920) noted the occurrence of severe purpura in fatal pneumococcal infection in children, and Bickel (1940) isolated the pneumococcus in a female, aged twenty-four years.

The occasional finding of the pneumococcus is of great interest, because of its ability to produce hæmorrhages.

Julianelle and Reimann (1927) established the fact that the pneumococcus extract with which they worked was thrombolytic both *in vivo* and *in vitro*, and that the development of purpura in mice following the injection of the extract was associated with an excessive diminution in platelets.

Mair (1928) confirmed the fact that pneumococci contained a substance which caused purpura in white mice, and also found that this substance was peculiar to the pneumococcus and did not occur in hæmolytic streptococci, *Streptococcus viridans* or diphtheria toxin, all of which gave negative results.

Lindsay *et alii* (1941) identified the bacteriological offender in four instances of the syndrome, two attributed to *Neisseria meningitidis*, two to *Hæmophilus influenzæ*, and one to *Neisseria flavus* II. Firor (1937) reports eleven cases of children with the Waterhouse-Friderichsen syndrome; three subjects died from staphylococcus septicæmia, one from erysipelas, one from *Streptococcus viridans* endocarditis, one from a streptococcal infection complicating a burn, one from bronchopneumonia, and one from an unexplained fever; but he does not mention whether purpura was present in every case.

In 1918 Winter had already drawn attention to post-mortem changes in the suprarenal glands in influenza.

Kellner (1933) reported the case of a child, aged five years, suffering from acute dysentery, in whom purpura was present before death, and thromboses of the suprarenal veins with widespread hæmorrhage into the gland were found post-mortem. He found hæmorrhage into the suprarenals to be a frequent complication of dysentery.

Lesions of the suprarenal glands can be found *post mortem* in patients who have died of many infectious diseases besides those already mentioned.

Ginandes and Howard (1947) note that the clinical picture may give no clue to the ætiology, and that the appreciation of the broad scope of bacterial agents which may produce the syndrome is important from the point of view of treatment.

Olitzki *et alii* (1942) showed that certain groups of micro-organisms had an adreno-hæmorrhagic effect when injected into guinea-pigs. Highly active strains were found in the families of the Enterobacteriaceæ, Neisseriaceæ and Pseudomonadaceæ. Weak irregular reactions were observed with some bacilli, staphylococci and streptococci. No reaction resulted from the injection of corynebacterium or mycobacterium. They noted that the reaction of the left suprarenal was frequently more distinct than that of the right.

Lithander (1945), in a most comprehensive publication, proved that, by the use of bacterial toxins, it was possible to produce the same clinical picture in animals as that called Waterhouse-Friderichsen syndrome in man. By comparison with adrenal insufficiency produced by adrenalectomy he made clear that the symptoms were due both to adrenal insufficiency and to the intoxication. The most prominent symptom, he thought, of failing or disturbed adrenal function was the derangement in carbohydrate metabolism. This surmise we now know to be correct.

Sachs (1937) suggested that the involvement of the skin and adrenal medulla, both of similar ectodermal origin, was evidence of the ectodermal tropism of the meningococcus; the pneumocóccus likewise possesses an ectodermal or epithelial tropism.

Some extremely interesting and instructive work has been done by Black-Schaffer and his colleagues (1947). These workers showed that washed meningococci, living or dead, were capable of producing the local Schwartzman phenomenon, and produced meningococcal purpura in rabbits, the lesions being interpreted as a generalized Schwartzman phenomenon. They were able to produce the Waterhouse-Friderichsen syndrome in the rabbit, and considered the adrenal lesions to be a by-product of the general toxæmia, and not necessarily the resultant of the Schwartzman reaction. They believe that the appearance of bilateral suprarenal cortical necrosis in experimental meningococcæmia is evidence of the ability of the washed bacteria to produce the Schwartzman reaction, and further suggest that this lesion is effected, not by thrombosis, but by vasospasm, with thrombosis as a sequel. These workers also emphasize the fact that the Schwartzman substance is not confined to the meningococcus, but is found, albeit in lesser concentration, in other organisms, and therefore it is not surprising that the Waterhouse-Friderichsen syndrome is occasionally produced by other bacteria.

Hopper and Schofield (1947) report the case of a white female, aged forty years, who had a bladder granuloma excised, and after operation went into a state of shock with cyanosis of the entire body (temperature 106° F.) and died rapidly. At the autopsy there were found generalized purpura of the skin, mucous membrane and serosal surfaces, hæmorrhagic necrosis of both suprarenals, diffuse necrosis of the bladder, acute splenic tumour (weight 925 grammes) and focal hepatic degeneration. No mention was made of the kidneys. The cause of death was an allergic angiitis with resulting purpura and hæmorrhagic necrosis of the adrenals. They believe that the patient was sensitized to products from the bladder, then received a "shock" dose of antigen as the result of surgery. They question the older conception of the bacterial nature of the Waterhouse-Friderichsen syndrome, and consider it best explained on the basis of tissue sensitization, an allergy of the vascular system.

A point of the utmost importance in this discussion is the question of the presence of lymphatic hypertrophy in this syndrome.

An enlarged thymus and/or hyperplastic lymph nodes have been observed in many instances. In the cases reported by Little (1901) the post-mortem notes remark on enlarged mesenteric glands and Peyer's patches, no special notice being taken of the thymus.

Westenhoffer (1905) wrote that in the German epidemic of spinal meningitis children with "lymphatic constitutions" were predisposed to infection with the meningococcus, and in 1923 Rabinowitz stated that the mesenteric glands were usually swollen and hyperæmic, and that the thymus might be enlarged.

McLean and Caffey (1931) stated that in seven of the subjects examined were found enlargement of the thymus and hyperplasia of lymphoid tissue, particularly of Peyer's patches and the lymphoid follicles. They considered that these were not border-line findings, but classical examples of what is termed *status thymolymphaticus*.

Baumann (1931) reported a large thymus and hyperplastic lymph nodes and follicles in his cases, and Magnusson (1934) found similar changes.

Bamatter (1934) discusses this question and draws attention to the close relationship between the thymus and suprarenals, and quotes Riedl, who stated that the *status thymolymphaticus* was connected with hypoplasia of the chromaffin system, and that, according to Wiesel and Hedinger, the under-development of the chromaffin system was the most frequent cause of thymic death.

In Aegerter's first case (1936) the thymus weighed 38 grammes, the subject being a seven years old child, and Sachs (1937) believes that the thymolymphatic prominence is an important factor in the pathogenesis of the disease.

Monford and Mehrling (1941) think that hyperplasia of the thymus and other lymphatic structures is associated with the Waterhouse-Friderichsen syndrome too frequently to be accidental, and Michael and Jacobus (1942), in their case, found the entire lymphatic system hyperplastic with areas of necrosis.

Martland (1944) found *status lymphaticus* of questionable degree in only three of 19 cases of the syndrome due to the meningococcus. The thymus in these cases was large, thick and fleshy, but the lymphoid tissue in the spleen and the intestinal tract was not excessive, and the mesenteric lymph nodes were not hyperplastic.

Schwartz (1946) found definite hyperplasia of the thymus and hypoplasia of the adrenal glands in five cases of the syndrome in children due to the meningococcus. He believes that there may be a predisposition in the thymo-lymphatic constitution of these children, which is also manifested in their pasty appearance.

Holmes and Cowan (1945), in two of three cases of this syndrome, found enlargement of the thymus and lymphatic glands; in one female child, aged eight years, the thymus weighed 41 grammes. They consider that, in view of the

short duration of the illness and the apparently normal histological appearance of the thymus and lymphatic glands, the enlargement must have existed previously, and it is possible that the adrenal hæmorrhage syndrome occurs only in individuals with an inherent structural and functional deviation from normal in their thymolymphatic system and adrenal glands.

Martland (1944), on the other hand, does not believe that there is any actual connexion between the *status thymolymphaticus* and the fulminating meningococcal infection.

No mention was made in the literature of any unusual changes in the spleen. Most authors, in autopsy reports, note that the spleen was enlarged. A small spleen, as in the case here reported, has not been previously found, and the possible relation of the abnormal spleen to the syndrome must be discussed. In our case these changes in the spleen must have been developing for years.

Congenital absence of the spleen is extremely rare, and from the meagre literature available it does not appear to be consistently associated with lymphatic hyperplasia.

Hodenpyl (1898), in an extremely interesting article, with a review of the old literature, reports the case of a coloured male, aged thirty-two years, who died of generalized tuberculosis with complete absence of the spleen, and in whom there appeared to be lymphatic hyperplasia of the lymph nodes. He quotes Stengel, who records the reported finding in an adult of a spleen weighing 3·63 grammes and measuring one and three-eighths inches by one-eighth of an inch by one-fifth of an inch.

McLean and Craig (1922) report a case of congenital absence of the spleen without lymphoid hyperplasia, and state that if lymphoid hyperplasia is in any way related to absence of the spleen it should be present in every case, and that from a review of the literature of this unusual anomaly one is justified in the conclusion that, in certain individuals, congenital absence of the spleen is apparently not a serious handicap.

Perla (1936) notes that removal of the hypophysis of the adult rat is followed by atrophy of the spleen. The administration of hypophyseal emulsion repaired the spleen to a considerable degree in such animals, and he suggested the presence of a spleen-stimulating factor in the anterior lobe of the hypophysis.

A relation of the hypophysis to the spleen was considered by Edwards and Wright (1937), who demonstrated that in splenectomized rats the pituitary gland increased in size and showed vascular proliferation, increase of the reticuloendothelial system, and increased mitosis of the basophilic cells.

Ungar (1945) found that in the course of the pituitary-adrenal reaction to stress this response was inhibited by splenectomy. He found and isolated the splenic extract which could reproduce the reaction to the original stimulus, and thought it was probably part of the anti-enzyme system which protects proteins against trypsin, and that the spleen, as an endocrine organ, might play a part in the control of protein metabolism and its adjustment to conditions of stress.

There are three reports in the literature of splenic atrophy resembling the changes in the spleen reported by us—Schilling's (1924), Grote and Fischer's (1929) (both of these being accompanied by atrophy of the remainder of the lymphocytic tissues), and that of Bigalke (1932), of splenic atrophy with severe anæmia.

In our case the atrophy of the spleen was associated with hypertrophy of the thymus and lymphatic glands, and its role in the fatal termination is unknown.

The ovaries were also affected. The pituitary was not examined.

Selye (1950) has enumerated many stressors which cause adrenocortical necrosis and hæmorrhages.

Considering all the findings, including the large thymus in Waterhouse-Friderichsen syndrome, Selye considers this syndrome to be a disease of adaptation. According to him, the derangement of the adaptive process appears to result from an exhaustive over-stimulation of the adrenal cortex with secondary acute hypocorticoidism.

We know with certainty that the lymphatic hyperplasia in our case was present before the fulminating terminal infection, and that the suprarenals could respond to stress, giving a normal response to adrenaline.

In Still's disease, with its splenomegaly, lymphocytosis and enlarged glands, we may postulate a moderate degree of *status thymolymphaticus*, which renders difficult any therapy against the arthritic manifestations. However, lately, in a few cases the condition has yielded to X-ray therapy with favourable joint response. In a number of cases of rheumatoid arthritis, both in children and in adults, there is evidence suggestive of an over-active thymo-lymphatic system. On the other hand, the enlarged lymph glands and spleen, with or without lymphocytosis, may be the expression of a subnormal suprarenal cortical activity.

In our patient suffering from Still's disease the allergic susceptibility may well have been the finger pointing to suprarenal cortical hypoactivity, and we might postulate that an over-active thymolymphatic system, by its anti-suprarenal cortical effect, lays the foundation for both allergy and fulminating infection. The literature which suggests that the association of the Waterhouse-Friderichsen syndrome and lymphatic hyperplasia is too common to be accidental, directs our attention to the necessity of recognizing such abnormality and of attempting to reduce susceptibility by X-ray irradiation of the thymus and mediastinal lymph glands.

Still's disease, always a difficult therapeutic problem, may be found to respond to such irradiation. Only time and observation can tell us the answer.

There is no doubt that early diagnosis of the Waterhouse-Friderichsen syndrome is essential, as now, with cortisone and antibiotics available, therapy is more likely to be successful. We can be certain that cortisone will revolutionize the therapy of the Waterhouse-Friderichsen syndrome.

Summary.

1. A fatal case is reported of the Waterhouse-Friderichsen syndrome due to the pneumococcus, in a girl who suffered from rheumatoid arthritis and who had given a normal response to Thorn's test.

2. Post-mortem examination revealed an enlarged thymus and extremely small spleen, with hæmorrhages into the suprarenals and ovaries.

3. The literature is reviewed, with special reference to the so-called *status thymolymphaticus* in this condition, Still's disease is shortly discussed, and a new line of treatment is suggested.

References.

ABGERTER, E. E. (1936), "The Waterhouse-Friderichsen Syndrome. A Review of the Literature and a Report of Two Cases", *J.A.M.A.*, 106: 1715.

ANLAYAN, A. J., KLIPPEL, C. H., and CURTIS, G. M. (1950), "The Waterhouse-Friderichsen Syndrome in the Differential Diagnosis of Acute Abdominal Crises", *Surgery*, 28: 763.

BAMATTAR, F. (1934), *"Fulminante Meningokokkensepsis. Zur Ätiologie des Syndrome von Waterhouse-Friderichsen"*, *Jahrbuch für Kinderheilkunde*, 142: 129.

BAUMANN, T. (1931), *"Zur Ätiologie und Klinik der akuten Nebennierenninsuffizienz (Nebennierenblutungen und -Hypoplasie)"*, *Ztschr. Kinderh.*, 51: 276.

BERNHARD, W. G., and JORDAN, A. C. (1944), "Purpuric Lesions in Meningococcic Infections. Diagnosis from Smears and Cultures of the Purpuric Lesions", *J. Lab. & Clin. Med.*, 29: 273.

BICKEL, G. (1940), *"Syndrome de Waterhouse-Friderichsen (septicémie suraiguë avec surrénalite hémorragique et purpura) terminé par la guérison"*, *Rev. méd. Suisse Rom.*, 60: 1053; quoted by Lithander, *loco citato*, 23.

BIGALKE, G. (1932), *"Über einen Fall von Milzatrophie mit einer Anämie perniziosähnlichen Charakters nebst Vergleich mit anderen in der deutschen medizinischen Literatur näher dargestellen Fällen von Milzatrophie"*, *Folia Hæmat.*, 48: 157.

BLACK-SCHAFFER, B., HIEBERT, T. G., and KERBY, G. P. (1947), "Experimental Study of Purpuric Meningococcemia in Relation to the Schwartzman Phenomenon", *Arch. Path.*, 43: 28.

DUDGEON, L. S. (1904), "The Etiology, Pathology and Diagnosis of Adrenal Hæmorrhage", *Am. J. M. Sc.*, 127: 134.

EDWARDS, L. T., and WRIGHT, C. W. (1937), "The Effect of Splenectomy on the Weight of the Hypophysis of the Albino Rat", *Endocrinology*, 21: 808.

FIROR, W. M. (1937), "Adrenal Hæmorrhage in Children", *South. M. J.*, 30 : 306.
FRIDERICHSEN, C. (1918), *"Nebennierenapoplexie bei kleinen Kindern", Jahrbuch für Kinderheilkunde*, 87 : 109.
GINANDES, C. J., and HOWARD, J. E. (1947), "A Case of Waterhouse-Friderichsen Syndrome due to Hemophilus Influenzæ", *J. Mt. Sinai Hosp.*, 14 : 778.
GLANZMANN, E. (1933), *"Beitrag zur Klinik, Hämatologie und Pathologie des Syndroms von Waterhouse-Friderichsen (Nebennierenapoplexie bei kleinen Kindern)", Jahrbuch für Kinderheilkunde*, 139 : 149.
GROTE, L. R., and FISCHER, B. (1929), *"Ueber totale Alymphozytose", München. med. Wchnschr.*, 76 : 2040.
HODENPYL, E. A. (1898), "Case of Apparent Absence of the Spleen with General Compensatory Lymphatic Hyperplasia", *M. Rec.*, 54 : 695.
HOLMES, J. M., and COWAN, J. M. (1945), "The Meningococcal-Adrenal Syndrome. Five Cases with One Recovery", *Lancet*, 1 : 13.
HOPPER, J. J., and SCHOFIELD, N. D. (1947), "The Waterhouse-Friderichsen Syndrome in Urology", *Urol. & Cutan. Rev.*, 51 : 509.
JULIANELLE, L. A., and REIMANN, H. A. (1927), "The Production of Purpura by Derivatives of the Pneumococcus. Eleven Further Studies on the Nature of the Purpura Producing Principle", *J. Exper. Med.*, 45 : 609.
KARSNER, H. T. (1942), "Human Pathology", 704.
KELLNER, B. (1933), *"Nebennierenblutungen bei Dysenterie", Zentralbl. allg. Path.*, 57 : 407.
KÖNIG (1922), *"Purpura fulminans bei einem 19monatigen Kinde", Ztschr. Kinderh.*, 32 : 282.
LINDSAY, J. W., RICE, E. C., SELINGER, M. A., and ROBINS, L. (1941), "The Waterhouse-Friderichsen Syndrome", *Am. J. M. Sc.*, 21 : 263.
LITHANDER, A. (1945), "Acute Adrenal Insufficiency in Rabbits Produced by some Bacterial Toxins", *Acta med. scandinav.*, Supplementum CLX, 1.
LITTLE, E. G. (1901), "Cases of Purpura Ending Fatally associated with Hæmorrhage into the Suprarenal Capsules", *Brit. J. Dermat.*, 13 : 445.
MAGNUSSAN, J. H. (1934), "Contribution to the Knowledge of Acute Suprarenal Insufficiency in Children (Suprarenal Apoplexy)", *Acta ped. scandinav.*, 15 : 396.
MAIR, W. (1928), "The Purpura Producing Substance in Pneumococci and the Heritable Susceptibility of Mice", *J. Path. & Bact.*, 31 : 215.
MARTLAND, H. S. (1944), "Fulminating Meningococcic Infection with Bilateral Massive Adrenal Hæmorrhage (The Waterhouse-Friderichsen Syndrome)", *Arch. Path.*, 37 : 147.
MCLEAN, S., and CAFFEY, J. (1931), "Epidemic Purpura. Meningococcus Bacteremia in Early Life. Diagnostic Values of Smears from Purpuric Lesion", *Am. J. Dis. Child.*, 42 : 1053.
MCLEAN, S., and CRAIG, H. R. (1922), "Congenital Absence of the Spleen", *Am. J. M. Sc.*, 164 : 703.
MICHAEL, P., and JABOBUS, L. (1942), "The Waterhouse-Friderichsen Syndrome", *Arch. Ped.*, 59 : 141.
MONFORD, J. A., and MEHRLING, J. H. (1941), "The Waterhouse-Friderichsen Syndrome", *Am. J. Dis. Child.*, 62 : 144.
NETTER, A., and SALANIER (1917), "The Presence of Meningococci in the Purpuric Elements of Meningococcal Infection", *Brit. J. Child. Dis.*, 14 : 101.
NOBECOURT, P., and MATHIEU, R. (1920), *"Le purpura dans les pneumococcies des nourissons", Arch. de méd. d. enf.*, 23 : 689.
OLITSKI, L., AVINERY, S., and KOCH, P. K. (1942), "The Hypothermic and Adreno-hemorrhagic Effects of Bacterial Vaccines", *J. Immunol.*, 45 : 227.
PERLA, D. (1936), "The Relation of the Hypophysis to the Spleen", *Am. J. Path.*, 12 : 792.
SACHS, M. S. (1937), "Fulminating Septicemia associated with Purpura and Bilateral Adrenal Hæmorrhage (Waterhouse-Friderichsen Syndrome)", *Ann. Int. Med.*, 10 : 1105.
SCHEIDEGGER, S. (1933), *"Ueber hämorrhagische Infarzierung beider Nebennieren bei Sepsis", Zentralbl. allg. Path.*, 57 : 163.
SCHILLING, V. (1924), *"Über die Diagnose einer Milzatrophie durch den Befund von Kernkugeln als Teilerscheinung pluriglandulärer Insuffizienz", Klin. Wchnschr.*, 3 : 1961.
SCHWARTZ, J. (1946), "Adrenal Hæmorrhages in Meningococcal Sepsis", *Arch. Path.*, 41 : 503.
SELYE, HANS (1950), "The Physiology and Pathology of Exposure to Stress: A Treatise Based on the Concepts of the General-Adaptation-Syndrome and the Diseases of Adaptation", Acta, Inc.
SNELLING, C. E., and ERB, T. H. (1935), "Hæmorrhage and Subsequent Calcification of the Adrenals", *J. Pediat.*, 6 : 22.
UNGAR, G. (1945), "Endocrine Function of the Spleen and its Participation in the Pituitary-Adrenal Response to Stress", *Endocrinology*, 37 : 329.
WATERHOUSE, R. (1911), "A Case of Suprarenal Apoplexy", *Lancet*, 1 : 577.
WEIL, M. P., and AZOULAY, R. (1924), *"Purpura aiguë staphylococcique", Bull. et mém. Soc. méd. hôp. Paris*, 48 : 258.
WESTENHOEFFER, M. (1905), *"Pathologische Anatomie und Infektionsweg bei der Genickstarre", Berl. klin. Wchnschr.*, 42 : 727.
WILLIAMS, H. (1942), "Meningococcal Infections in Infancy and Childhood, Part II. Meningococcal Septicæmia with Special Reference to Adrenal Apoplexy or the Waterhouse-Friderichsen Syndrome", *M. J. Australia*, 2 : 557.
WINTER, E. S. (1918), "The Suprarenal Glands in Influenza", *Brit. M. J.*, 2 : 629.

Reviews.

History of the Second World War: United Kingdom Medical Series. Editor-in-Chief, Arthur S. MacNalty, K.C.B., M.D., F.R.C.P., F.R.C.S. "Medicine and Pathology", edited by V. Zachary Cope, B.A., M.D., M.S. (London), F.R.C.S. (England), with a general preface to the series by the Editor-in-Chief; Volume I; 1952. London: Her Majesty's Stationery Office. 9¼" × 6¼", pp. 594, with one illustration and two text figures. Price: 50s.

REFERENCE is made in the leading article on page 307 to the United Kingdom Medical Series of the History of the Second World War and to the volume on medicine and pathology which has been edited by Sir Zachary Cope. Readers will learn from the leading article something about the whole series and also about this first volume. It remains only to indicate in a general way the subjects which are dealt with in this volume and to commend it to the attention of readers.

There are in all 33 chapters, and some of them are divided into several sections. Most of the chapters have been contributed by authors who are not members of the editorial staff, but in one or two instances the chapters are marked as having been edited by Sir Zachary Cope or as having been collated by him from various contributions.

The first chapter, in some ways the most important in the volume, is divided into four sections dealing respectively with medicine in the Royal Navy, medicine in the Army, medicine in the Royal Air Force, and medicine in the Emergency Medical Services. The authors of these four sections are Sir J. W. McNee, Brigadier John Bennet, Sir John Conybeare and Sir Arthur S. MacNalty in collaboration with Professor O. L. V. deWesselow. In the section dealing with the Navy, reference is made to the development of convoy rescue ships, which were small converted merchant ships equipped with an operating theatre, a small hospital and many bunks for survivors. These ships saved thousands of lives. To show the endurance of some of the survivors, we feel compelled to include the history of a stoker petty officer who was sleeping over the engine room in a ship that was torpedoed in the engine room. He had second degree scalds on the face, trunk and arms, and third degree scalds on his hands. He was blown into the sea which was at a temperature of 29° F. and swam nearly a mile to a trawler. Three or four hours later he was transferred to a convoy rescue ship, where he was treated for shock by plasma transfusion and other means. He made a good recovery and in due course was able to return to duty. In the section dealing with the Army, the incidence of disease and wastage are described and the various types of diseases are discussed. Under the heading of "Some Lessons Learned" mention is made of what must be learned for any future war. Technical advances have led to enormous reduction in wastage, and we read that as the machinery of war becomes more complex and the personnel engaged in the field and the factories become more highly trained, wastage matters more and its prevention takes on added importance. In this matter, priority has to be given to measures of prevention of disease applied at the source. The foundation of success at this level in the Army is "good man management and military discipline". Emphasis is laid on the role of the regimental medical officer. An efficient "R.M.O." is "a specialist in man power conservation". We are told that he "should have energy and initiative, a personality which will ensure within reason that his necessarily tireless propaganda on matters of health discipline is accepted with conviction, and a sound knowledge of preventive medicine and the maintenance of morale in all their aspects". Those who served in the First and Second World Wars know that he often did have all these qualities. Reference is also made to a spirit of cooperation which was apparent at an international level, and by means of which observations on obscure conditions in different armies were collated and given added form until completeness was reached.

In the section dealing with the Royal Air Force a good deal of space is devoted to cardio-vascular problems. The diagnosis of effort syndrome was virtually absent from the Royal Air Force when there was every reason to expect that many cases of the condition would occur. This absence is attributed to the occurrence of fashions in diagnosis, and it is suggested that the absence of effort syndrome was primarily due to the use of other diagnoses to describe it. It is noted that officers of particular value and experience who had had coronary occlusions were often retained in the service, and the conditions under which this was done are described.

In the section devoted to medicine in the Emergency Medical Services, we read that from a heterogeneous collec-

tion of over 3000 medical institutions of various grades under a variety of systems of administration, upwards of 1000 hospitals, well equipped and as efficiently staffed as the shortage of man and woman power would permit, were welded into a homogeneous hospital service. These participating hospitals, in addition to their normal obligations which had prevailed in peace time, were required to treat all patients, civil and military, for whom the Minister of Health had accepted responsibility, and to transfer patients to other hospitals in the service in order that the necessary number of beds might be available for new admissions in the areas where they were required.

The chapter on dyspepsia is full of interest. Various kinds are described, and among them are acclimatization dyspepsia, reflex dyspepsia, migrainous dyspepsia, psychogenic dyspepsia, and emotional dyspepsia. When the tide began to turn and victory approached, there was a rapid improvement in morale; the service population began to diminish rapidly and the problem of dyspepsia faded into comparative insignificance. The same kind of experience was reported in Germany—"that dyspepsia was a product of static war and that during active warfare it faded away". Between the wars a large ulcer population grew up, and when these persons were taken away from their accustomed occupation, put into uniform and deprived of their careful diet, so many of them developed recurrences that there arose for the service authorities a problem of the first magnitude. The malaria problem is discussed by Colonel J. S. K. Boyd in a chapter on advances in tropical medicine. He points out that in the past, malaria was shown to be the greatest source of man power wastage in regions where it was endemic, and explains that it was in Burma and the South-West Pacific that malaria appeared in its most serious form and for a time reached endemic level. He describes the work carried out at the Land Headquarters Medical Research Unit of the Australian Imperial Force at Cairns, the unit which, as A. S. Walker explains in his volume of the Australian history, owed its original conception to H. K. Ward, E. V. Keogh and I. M. Mackerras. It was N. Hamilton Fairley who organized the unit and was chiefly responsible for its success. Boyd gives only a summary of the findings of the unit and adds in a footnote that a full account of its work will be found in the first volume of the Australian Medical History. Bennet, in his chapter on medicine in the Army, states that of all the achievements of medical research dictated by the war, the control of malaria was perhaps the most outstanding. He thinks that its pride of place can be disputed only by any corresponding impetus given to the production of penicillin. He also adds that deaths from malaria virtually disappeared as a cause of wastage.

In discussing the typhus fevers, C. H. Andrewes refers to the Typhus Research Committee which was appointed by the Medical Research Council at the request of the War Office. He discusses four aspects of typhus, vaccine production and testing, serology and antigenic structure of rickettsiæ, chemotherapy and insecticides. In his discussion of mite-borne or scrub typhus, he refers to the work of R. N. McCulloch on dimethylphthalate and dibutylphthalate.

At the end of his discussion on infective hepatitis, E. R. Cullinan concludes that in spite of all our added knowledge, the epidemiological puzzle of the condition remains unsolved —many pieces of the jigsaw have been fitted into place, but the key pieces are still missing.

Neurology is dealt with in two sections—the first, neurology proper, and the second, nutritional disorders of the nervous system.

The chapter on psychological medicine is a large one. After a general introduction, psychiatry in the Navy is discussed by Desmond Curran, psychiatry in the Army is discussed by Brigadier R. J. Rosie, and psychiatry in the Middle East Force by G. W. B. James. The section on neuropsychiatry in the Royal Air Force is edited by Sir Zachary Cope and based on the work of Symonds and Williams. Psychiatry in the Emergency Medical Services is dealt with by Aubrey Lewis and Eliot Slater. No less than 118,000 men and women were discharged from the services for psychiatric reasons between September, 1939, and June, 1944. Between one-third and one-half of all the medical invalids, men and women, discharged from the services were discharged on psychiatric grounds. This represents from four to ten per thousand of the average strength *per annum*. Of the 118,000 persons discharged on psychiatric grounds, 64·3% had a diagnosis of psychoneurosis and effort syndrome (Sir John Conybeare, we noted previously, has stated that this diagnosis was not used), 21% had a diagnosis of psychosis, 6·4% a diagnosis of mental defect, and 8·1% had a psychopathic personality. Looking at this enormous total, we can imagine what a great deal of time was taken up in the examination, treatment and final disposal of these persons. It is interesting to note that during their training, army psychiatrists were posted to a field force to live for four weeks in the atmosphere of fighting units. During this period, they acted neither as officers nor as psychiatrists, but were observers of, and participants in, the unit's activities.

Among the other chapters which must be passed over for lack of space are those on the results of exposure after shipwreck and the like, on tuberculosis in the Royal Navy, and on the hazards of toxic gases in warships apart from agents of chemical warfare. It may be surprising to some persons to find a chapter on pædiatrics during the war. In this, the late Sir Leonard Parsons discusses by-products of war conditions, the influence of diet on mother and offspring, the causes of deaths among children, deficiency diseases and other morbid conditions in war time. The other chapters need not be named; no subject seems to have been omitted.

One defect in the volume, which is much to be regretted, is the absence of uniformity in the matter of printing the references. In the preface Sir Zachary Cope states that he would have liked to adopt the well-known Harvard system, but apparently he gave way to the preferences of authors and states that it was not possible to achieve uniformity. It would have been an advantage if the titles of articles had been included, regardless of the adoption or non-adoption of the Harvard system. The one or two instances in which this has been done serve only to show up the very many in which no titles are given.

In commending this volume to the careful attention of Australian practitioners, we may conclude with a quotation from the contribution by Sir Arthur McNalty on medicine in the Emergency Medical Services:

> In the face of countless difficulties, British Medicine, as the chapters in this volume reveal, was not only maintained at a high level during the war, but medical research increased its armamentarium and enabled physicians to treat effectively some diseases which had been regarded as invariably fatal. . . .

Massage and Remedial Exercises: In Medical and Surgical Conditions. By Noël M. Tidy, T.M.M.G.; Ninth Edition; 1952. Bristol: John Wright and Sons, Limited. 9″ × 6″, pp. 528, with 192 illustrations. Price: 27s. 6d.

THE ninth edition of "Massage and Remedial Exercises", by Noel M. Tidy appearing only three years after the previous edition, proves the continued popularity of this work. It contains physiotherapeutic details for diseases of all the bodily systems and for both general medical and surgical maladies as well as the purely orthopædic ones. Also a series of exercises for the pre-natal and post-natal periods are described and even exercises to help keep the aged fit as well as those for the treatment of obesity and constipation.

Some 139 pages are devoted to fractures and bone and joint diseases and all phases of orthopædic physiotherapy are detailed. Whether the reader is a practising physician or surgeon or a physiotherapist, aspects of remedial massage are enumerated that will help in the treatment of all diseases in which massage can possibly be used; all are designed to help patients to return to useful activity in as short a space of time as possible and so to relieve the tedium of a long spell in bed.

Books Received.

[*The mention of a book in this column does not imply that no review will appear in a subsequent issue.*]

"Aids to Public Health", by Llywelyn Roberts, M.D. (Hygiene), M.R.C.P., D.P.H.; Seventh Edition; 1952. London: Baillière, Tindall and Cox. 6½″ × 4″, pp. 328, with five text figures. Price: 7s. 6d.

One of the well-known "Students' Aids Series".

"Human Parasites and Parasitic Diseases: For Students, Laboratory Workers, Practitioners of Medicine and Public Health", by K. D. Chatterjee, M.D. (Cal.); 1952. Calcutta: K. D. Chatterjee. 10″ × 7½″, pp. 774, with 328 illustrations, 82 in colour. Price: £6.

Intended to satisfy the needs of parasitologists, clinicians, laboratory workers and research workers.

The Medical Journal of Australia

SATURDAY, FEBRUARY 28, 1953.

All articles submitted for publication in this journal should not be sent. Authors are requested to avoid the use of abbreviations and not to underline either words or phrases.

References to articles and books should be carefully checked. In a reference the following information should be given without abbreviation: surname of author, initials of author, year, full title of article, name of journal without abbreviation, volume, number of first page of the article. If a reference is made to an abstract of a paper, the name of the original journal, together with that of the journal in which the abstract has appeared, should be given with full date in each instance

Authors who are not accustomed to preparing drawings or photographic prints for reproduction are invited to seek the advice of the Editor.

THE BRITISH MEDICAL HISTORY OF THE WAR.

THE official medical history of the Second World War for the United Kingdom, as opposed to the histories to be produced by the other units of the British Commonwealth, is being published under the title "History of the Second World War: United Kingdom Medical Series". The Editor-in-Chief is Sir Arthur S. MacNalty, sometime Chief Medical Officer of the Ministry of Health. A splendid organization was built up for the production of the history. First of all a War Cabinet Committee for the Control of Official Histories was set up; this afterwards changed its name and dropped the word "War" from its title. It was resolved that an official medical history should be part of the committee's plan, and it is to be noted that the War Cabinet laid it down that the history should be on a coordinated basis and should include not only the medical side of the three fighting services, but also the medical civilian services. An Editorial Board was appointed, composed of service and departmental representatives. On the board were the directors-general of various services and they appointed medical representatives to collect material for the history; these representatives were subsequently called service editors. The directors-general were regarded as supervising editors. The Editorial Board which meets twice a year appointed an Editorial Committee which comprises the Editor-in-Chief with representatives from the Admiralty, the War Office, the Air Ministry, the Board of Education, the Ministry of Pensions, the Department of Health for Scotland, the Medical Research Council and a secretary. At headquarters where the work was done the Editor-in-Chief has with him the secretary and three editors—Lieutenant-Colonel C. L. Dunn (formerly of the Indian Medical Service), Dr. N. G. Horner (formerly Editor of the *British Medical Journal*), and Sir Zachary Cope, F.R.C.S. The enormous amount of work that these people had to do may be easily imagined and one may be forgiven for wondering whether they did not at times find the top-heavy organization behind, or shall we say above, them a burden rather than a help. One aspect of the organization which is to be highly commended is the establishment of close collaboration between the medical historians of the several countries in the British Commonwealth and the United States of America. In 1946 a permanent Official Medical Historians Liaison Committee of the Commonwealth countries was set up and conferences have been held at Ottawa in 1947, Oxford in 1948, Canberra in 1949 and Delhi in 1952. The countries represented were: Australia (Dr. A. S. Walker), Canada (two representatives), India and Pakistan (two representatives), New Zealand (Dr. T. D. M. Stout), South Africa (Dr. C. C. P. Anning), United Kingdom (six representatives, including Sir Arthur MacNalty and Sir Zachary Cope); three representatives attended from the United States as observers. The results of the meetings of this committee are reflected at several points in the first volume of the United Kingdom series.

The first volume, entitled "Medicine and Pathology", edited by Sir Zachary Cope, which has recently been published, will be followed by others. The other subjects are as follows: "The Civilian Health and Medical Services"; "The Emergency Medical Services"; "Surgery"; "The Naval Medical Services: (*a*) Administration, (*b*) The Campaigns"; "The Army Medical Services: (*a*) Administration, (*b*) The Campaigns"; "The Royal Air Force Medical Services: (*a*) Administration, (*b*) Commands, (*c*) Campaigns"; "Medical Research"; "Medical Statistics". Clearly the whole range of subjects connected with war will be covered. In regard to technical subjects the Editor-in-Chief writes that the cover will include the whole field of scientific and practical advances in every subject which has a bearing on disease and injuries as they occur in wartime. Place will even be found for such subjects as wartime medical education, the social aspects of the war in relation to disease, and the rehabilitation of the sick and wounded. In these circumstances we may expect that the clinical volumes of the British history will deal, as the Editor-in-Chief declares that they will, "with those problems of injury and disease which confronted medical officers in the various theatres of war" and that they will "record the many advances in our knowledge and the application of this knowledge to the prophylaxis and treatment of disease and in countering the results of injury".

We do not propose to deal in this place with the details of the contents of this book, except to point out that they comprise thirty-three chapters (some divided into sections) contributed by various authors—they are described elsewhere in this issue. What we may consider is the purpose and general value of such productions as medical histories. The history of a war as it affects the existence—the well-being or the adversity—of a nation will be regarded by everybody as worthy of study or at least of attention. And wars are sometimes made the chief features of history as taught to school children. In our recent presentation of the first volume of Dr. A. S. Walker's Australian medical war history the historical method was mentioned and it was pointed out that it was a complicated process. It is useless to consider the history of war and its effects apart from the medical component. The importance of the medical services can be looked at from several points of view. One of the most significant is that with the

present-day efficiency of army medical service the morale of the troops is lifted—the soldier realizes that unless he receives a fatal wound he will, in a reasonably short space of time, be brought to a place where he can receive really adequate medical attention. The aeroplane is the main factor in this. In comparison with the First World War, the Second was a war of movement and casualties had to be, and were, moved quickly. Air services were often used. The war in Korea has added another useful agency —the helicopter. It has been said that the one thing we learn from the study of history is that we do not learn from its study. (Sir Henry Tidy, in the introduction to the first volume, uses a variation of this, to the effect that there is no lesson to be learned from it—this is obviously incorrect.) The saying quoted by us is not quite true, though there is a certain amount of truth in it. What probably happens is that at the outbreak of war there is an attempt to apply the methods used with success at a previous conflict without the paying of attention to the fact that the type of warfare has changed. There is an enormous difference, for example, between the trench warfare of the First World War on the one hand and the desert and jungle types of warfare of the Second World War on the other. Organization, of course, comes into the picture and will be set up at the outset of a war to meet existing conditions as far as possible; we may also be quite certain that organization will improve as the period of warfare becomes longer. It is very easy at the beginning of a war, or indeed at any stage, to attribute deficiencies of administration, apparent or real, to unwillingness to apply lessons previously learned. Looking back to the end of the First World War, we may reflect that the Spanish Civil War gave some new methods to military medicine and we have already remarked that the Korean War has not been unprofitable to army medical methods. When all is said and done most attention has to be paid and most credit has to be given to advances that have been made in medical science. This first volume of the British history shows what these were. One thing is clear and that is that the combatant arms of the services and those who control the general conduct of war have had demonstrated to them, and realize, the enormous contribution which the medical services can and do make. We may also take it that the value of research in war medicine is firmly established. We shall soon have in print the experience of all the countries of the British Commonwealth in the Second World War. If the disaster of a third world conflict should overtake humanity, conditions will be very different from anything previously known, and it is surely reasonable to suggest that research should be undertaken or preparations made against that great and terrible day. But that is for discussion on another occasion.

Current Comment.

BODY ARMOUR IN WARFARE.

Body armour for soldiers in battle went out of fashion several centuries ago, but by a curious turn of events it is again coming into prominence. Advances in the evacuation and treatment of the wounded have so reduced the mortality amongst those who have not been fatally wounded in the first place that for further reduction in the over-all mortality of warfare it would seem necessary to look for protection from wounding itself. Lieutenant-Colonel R. H. Holmes,[1] in a paper read before the Section of Military Medicine at the American Medical Association's Annual Session in June, 1952, states that the mortality amongst those wounded in action has dropped to 23 per 1000 in Korea as compared with 45 per 1000 in World War II. No doubt this rate can be still further reduced with technical advances in treatment, but not to any great extent; further significant reduction must come, as Holmes points out, from the group that die before admission to hospital. The possible means of accomplishing this are prevention of wounds, reduction in number and severity of wounds, and faster recovery from the battlefield of those wounded. The helicopter has contributed strikingly to the last-mentioned of these. For the other two it seems that we must look to some sort of body armour. The interesting point has been made by Brigadier-General A. F. U. Green[2] that the death knell of armour of the earlier type was due to increasing weight, complexity and cost and not to the use of firearms. Indeed, the bow and arrow had remarkable penetrating power, which was often combined with great skill and accuracy. Green states that the long bow, used in thirteenth century England, could penetrate an inch of oak at 200 yards, and it was reckoned that an archer should hit a target the size of a man's head and trunk at any range up to 100 yards almost without fail. Primitive firearms, on the other hand, could kill or maim only by concussion. Armour had vanished from the battlefield well before the firearm established superiority over the bow or crossbow around the end of the seventeenth century. The impracticability of continuing the use of armour is obvious from Green's estimate that a knight's horse was expected to carry over three hundredweight, made up as follows: helmet, 25 pounds; armour and weapons, 80 pounds; horse furniture, 80 pounds; man, about 150 pounds; sundries, 15 pounds. Various half-hearted attempts have been made since to use the principle of armour, including experiments by both Germans and Allies in World War I, according to Holmes. The "tin hat" was the only significant result. Holmes states that towards the end of World War II a fairly large number of thoraco-abdominal vests were made by the United States forces but never satisfactorily tried on the field of battle; the use of body armour had been standardized by the Air Force among aircraft personnel, for whom weight of the protective item was less important than it was for field troops. Considerable stress has been laid on keeping the weight of the infantryman's equipment to a minimum, and this must be remembered always in relation to body armour. The other factor is the nature and site of wounds produced by modern weapons. Wound ballistics studies conducted under the direction of the Surgeon-General, Department of the Army in the United States, have established the anatomical regional frequency and distribution of wounds, the relative incidence of wounding agents, the circumstances of wounding and a classification of the type of wound produced; on the basis of such studies the feasibility of body armour has been proposed. Wounds to the thorax account for nearly as many of those killed in action as head wounds. Thoracic and abdominal wounds account for one-third or more of those killed in action. Shell fragments cause about 75% of all wounds and 50% of deaths of those killed in action. The sites and types of a majority of wounds suggest the possible value of body armour, and an effort has been made to provide it. The first controlled field trial of body armour was made in 1951 in Korea with a thoraco-abdominal vest weighing less than eight pounds; this is possibly the same jacket as the one described by Green as consisting of "Nylon" backed by sponge rubber. He refers also to another jacket made of laminated plastic fibre glass and weighing seven and three-quarter pounds. Holmes states that after a field trial of two months' duration, it was reported that the vest was desirable and acceptable and could be worn without undue encumbrance or hindrance. The psycho-

[1] *J.A.M.A.*, September 13, 1952.
[2] *United Service*, January, 1953.

logical effect was also favourable. Further trials with modified garments are still going on, and the results are stated to be encouraging. Many practical aspects will, of course, need to be considered with great care, especially the viewpoint of the men who have to wear the armour. However, the idea is worthy of the closest investigation, especially as modern synthetics such as "Nylon" and compressed fibre glass give us great advantages in the way of materials over the knights of old.

AMERICAN MEDICAL STUDENTS ABROAD.

WE are accustomed to Australians going abroad for post-graduate experience and study, with and without the intention of collecting higher degrees and diplomas. The development of post-graduate teaching in Australia and the establishment here of higher degrees and diplomas of excellent standard have removed some of the reasons for this; but the value of contact with those working in the major clinical and research centres of Britain, some parts of the Continent and America is obvious, and Australians will rightly continue to draw on what these countries have to give, while realizing that Australia is assuming increasing importance to students from Asia. It is unusual, however, for many Australians to seek undergraduate training abroad, though there may be some who think that Europe and North America have more to offer than Australia's four medical schools. Curiously enough a survey conducted in 1951, according to F. R. Manlove,[1] revealed that at least 1121 American students (we take it that this means students from the United States) were enrolled in foreign medical schools. The greatest number (363) were in Switzerland. All the 222 in Spain were from Puerto Rico, as were 54 of the 68 in Mexico, which is not surprising when we recall that Puerto Rico was ceded to the United States by Spain only a little over half a century ago. The other comparatively large enrolment was in Italy (134), and it is thought that the true figure is probably much higher. The Netherlands had 74, Germany 62, France 44, Ireland 29, Cuba 27, Australia 24, Austria 24, Great Britain 17 and Belgium 11. It is interesting to note that the Americans made up a substantial proportion of students in some European universities (43·8% of the total student body at Fribourg, 45·3% of two classes at Berne and 20·6% of all medical students at Geneva). The reasons for the exodus are not always clear, though undoubtedly some of the students were unable to gain admission to approved American medical schools. There is apparently keen competition to enter these schools. The fifty-second annual report on medical education in the United States and Canada by the Council on Medical Education and Hospitals of the American Medical Association[2] lists 72 medical schools in the United States and 10 in Canada approved by the Council, as well as eight approved schools of the basic medical sciences in the United States and Canada; the total enrolment for these 90 schools was 29,680. This makes the 1121 medical students in foreign schools a comparatively small band, but the figure is not negligible, especially as most medical schools are, like our own, struggling with greater numbers than they care to have. It may be noted that of the 24 American students in Australian medical schools 18 were in Melbourne. An Australian school (unidentified) stated on inquiry that American students were badly prepared, but attributed this to the fact that only second rate American students had applied for admission. Inquiry elsewhere was not always very enlightening. "Most comments indicated that the American students were diligent, their conduct satisfactory, and their academic performance equal to that of the average students", though Manlove does not consider that the last comment could be construed as complimentary, at least in some European schools. An Irish school found the Americans superior to native students: a Dutch school rated them worse. One foreign university official stated that schools in his country wanted American students; they did not expect to get "the cream" of the American pre-medical group, but hoped to get "some of the milk"; two schools apparently felt that they had been getting "skimmed milk—and a bit curdled at that". Certainly, despite a proportion of good students, it seems that difficulty of inferior students in gaining admission to approved American medical schools offers the main explanation of this rather curious phenomenon. An interesting side issue mentioned by Manlove is the difficult task that will be faced by licensing authorities in the United States when they attempt to evaluate medical graduates returning home from 21 different countries.

[1] *J.A.M.A.*, September 13, 1952.
[2] *Ibidem.*

THE MANAGEMENT OF CŒLIAC DISEASE.

AFTER many years of trial-and-error dietetic treatment of children suffering from cœliac disease, a notable advance was made with the clear incrimination of gluten. Why gluten, even in small quantities, is harmful to these children is not precisely known yet, but, as Wilfrid Sheldon and David Lawson[1] state, the present position concerning our dietetic management of children with cœliac disease may be summed up by saying that the objective is a diet from which gluten is completely excluded, but which is in other respects normal. The importance of excluding gluten from the diet and the light that this throws on previous experiences of dietetic therapy were discussed in these columns on June 21, 1952, particularly in relation to the work of Charlotte Anderson, Professor A. C. Frazer and their colleagues at Birmingham. Sheldon and Lawson's article, based on experience in the treatment of 64 children suffering from cœliac disease, deals with the practical aspects of treatment in detail and merits the closest attention. These main points are brought out: first, that gluten must be completely excluded from the diet; second, that the reintroduction of gluten requires the greatest care and may well be delayed for a long period; third, that the removal of gluten, essential though it is, does not complete the dietetic management of a child suffering from cœliac disease. Sheldon and Lawson state that they have made no precise experiments to determine the "effective threshold toxic dose" of wheat gluten, but it is within their experience that this is small. They have observed that one slice of toast a day (containing two grammes of gluten) can check growth. With regard to the reintroduction of gluten to the diet, they emphasize the point that to base a return to gluten simply on a clinical assessment of well-being is insufficient. "All too often, when gluten is reintroduced the rate of growth promptly slows, although if treatment has persisted for several months the complete picture of cœliac disease does not necessarily reappear." This interference with growth can be overlooked if a careful and regular check is not being made and especially if the attending doctor has not accepted and absorbed Sheldon and Lawson's dictum that there is no justification now for accepting anything short of optimal growth as evidence of success in treatment. They suggest that it may be necessary to withhold gluten throughout the period of growth of the child, but are not yet in a position to say that this is so. Dealing with other aspects of the dietetic regime, they point out that care, experience and persuasion may be needed to institute the gluten-free diet, that the diet must be satisfactory in other respects and especially must provide enough calories, and that anæmia and infection require particular attention. On the practical details of the diet, not only as it is arranged in hospital but also as it offers problems to the mother at home when the child has left hospital, this paper is full of useful and thoughtful advice. Some of the detail about rationing is irrelevant in Australia, and some of the special food products mentioned may, unfortunately, not be available here. The article, however, covers the practical aspects of the subject well and can be studied with advantage by everyone who has to treat a child with cœliac disease. There seems little excuse nowadays for these children to do other than very well.

[1] *Lancet*, November 8, 1952.

Abstracts from Medical Literature.

THERAPEUTICS.

The Non-Rebreathing Method in Pædiatric Anæsthesia.

C. R. STEPHEN (*Anesthesiology*, January, 1952) emphasizes some important physiological phenomena in pædiatric anæsthesia and discusses the recognized techniques used. He states that obstruction of the airway must be avoided at all times, and if the usual methods are not satisfactory one should be ready to insert an endotracheal tube. Anoxia, excessive dead space, accumulation of carbon dioxide and resistance in the apparatus should be eliminated. With the open-drop technique with ether, the diminution in the oxygen tension, increase in the carbon dioxide tension and dead space under the mask can be reduced by giving half to one litre of oxygen under the mask, but other disadvantages are that only one agent can be used and there is no means of artificial assistance to depressed respiration. In the partial rebreathing (Boyle's) technique commonly used in Great Britain, which involves a big flow of gases and an expiratory valve on the face mask, there is little resistance; but accumulation of carbon dioxide can occur, and there is considerable dead space in the face mask. In cycle circuits there is too much resistance for children under eight years and in to-and-fro systems for children under two years. With any rebreathing technique the respirations should be assisted through the entire anæsthetic administration. Combinations of partial rebreathing technique and to-and-fro absorption systems are satisfactory, especially if an endotracheal tube is used. Ayre's "T"-tube with insufflation of the anæsthetic gases at relatively high rates is advantageous, in that dead space and resistance are minimal; but constant insufflation is not physiological and may lead to carbon dioxide excess, and also there is no rebreathing bag to control the respiration correctly. The most recent method, the non-rebreathing technique, entails the use of very light rubber flaps, which act as valves deviating the entire exhalations into the air. The resistance in this circuit has been shown to be 1·75 centimetres of water on the inspiratory side and 1·0 centimetre on the expiratory side. The valve is connected to the endotracheal tube, and a rebreathing bag also is connected which can be used for controlled respiration after the thumb is placed over the exhalation valve. Of the various techniques which have been enumerated, the author states that the non-breathing method satisfies best the demands made of the anæsthetist in pædiatric anæsthesia.

Pressor Amines and Shock from Myocardial Infarction.

H. K. HELLERSTEIN, B. L. BROFMAN AND W. H. CASKEY (*Am. Heart J.*, September, 1952) report the treatment with pressor amines of shock accompanying myocardial infarction, a condition with an associated mortality of 80%. Of 18 patients, 17 were treated with "Mephentermine" (three also receiving ephedrine) and one with ephedrine alone. Shock was considered to be present when hypotension (a systolic blood pressure of less than 90 millimetres of mercury in previously normotensive patients) persisted for more than one hour, and when the classical signs and symptoms of circulatory collapse were present. The pressor substance was administered intravenously in five to 20 milligramme doses or by slow intravenous drip of approximately one milligramme per minute until a sufficient pressor response was produced. In a few cases, the intramuscular route was used in doses of 35 milligrammes. It was deemed advisable not to produce a great pressor response, and usually an attempt was made to maintain the systemic systolic blood pressure at approximately 100 millimetres of mercury. Generally, the pressor drug had to be given again over a period of one to ten hours before the blood pressure was maintained. The authors state that the aim of therapy in shock accompanying myocardial infarction is to restore the effective head of blood pressure in the aorta so as to perfuse adequately the coronary, cerebral, renal, hepatic and other vital circulations. Measures designed to combat shock are directed toward supporting the uninfarcted muscle. Elevation of the systolic blood pressure (to 100 millimetres of mercury in patients previously normotensive, and 120 millimetres of mercury in those previously hypertensive) improves the total coronary circulation, enhances the chances of survival of borderline myocardium and that supplied by stenotic arteries, and may decrease the size of the infarct. The ideal pressor drug would elevate blood pressure, increase peripheral resistance, produce a proportionate increase of coronary flow, have minimal side effects and not decrease cardiac output or produce serious arrhythmias. Some amines now available fulfil most of these requirements, and "Mephentermine" was regarded by the authors as the amine of choice in this study. It was found to be a safe, effective pressor substance with little direct effect upon myocardial irritability in the doses employed. A pressor response was produced in 16 of the 18 patients; 14 emerged from the shock state with considerable clinical improvement for more than two days. However, seven succumbed to secondary complications two to twenty-six days later. Seven patients recovered sufficiently to be discharged from hospital. Pressor therapy did not produce congestive failure in patients not previously in a state of failure, or aggravate preexisting failure.

Stellate Block for Bell's Palsy.

DANIEL M. SWAN (*J.A.M.A.*, September 6, 1952) presents the clinical history of two patients suffering with Bell's palsy who were treated by stellate block, in one case five times within seven days, and in the other four times within six days. Procaine hydrochloride was introduced by the anterior method of approach on the homolateral side. The causes of Bell's palsy are listed as idiopathic 87·2%, trauma 6%, infection 6%, and tumour 0·8%. The author states that although Bell's palsy is not a serious disease and recovery is spontaneous in most cases, this recovery is often long delayed with much associated psychological distress. The author's two patients had palsy of the idiopathic type, and the treatment resulted in return of function more rapidly than could have been expected in the natural course of the disease, even with the application of the best previously known therapy. The author suggests that his results tend to confirm Kettel's theory of a primary "dysregulation" of the circulation involving the *vasa nervorum* as the primary ætiological factor in the idiopathic type of this disease. This "dysregulation" would presumably be of a vasospastic nature producing ischæmia and resulting malfunction of the nerve. The author recommends this method of repeated stellate ganglion block with procaine hydrochloride, carried out on the homolateral side, for patients with the idiopathic type of Bell's palsy, the treatment being preferably instituted early in the course of the disease.

Pneumococcal Meningitis.

P. A. BUNN AND G. PEABODY (*Arch. Int. Med.*, May, 1952) describe the treatment with penicillin of 20 patients suffering from pneumococcal meningitis. Intrathecal penicillin therapy was not considered desirable on account of poor penetration and irritative effects. Administration of 1,000,000 units of penicillin every two hours by the parenteral route gave the best results. Of 20 patients treated, five died and 15 survived. Sulphonamides and aureomycin did not help.

Hypertension.

M. MOSER *et alii* (*Arch. Int. Med.*, May, 1952) describe the results of blockade of the sympathetic nervous system by chemical means in cases of hypertension. They state that thiocyanates were toxic and *veratrum viride* was variable in effect. Barbiturates were helpful, nitrites were ineffective. "Priscoline", "Regitine", and dihydroergocornine also had variable effects. "Dibenamine" and its derivative 688-A-N-pyenoxyisopropyl-N-benzyl-β-chloroethylamine hydrochloride were effective, the latter being less toxic and effective when given orally. Eleven patients were treated. Diastolic blood pressure fell in five cases when the patients were in the recumbent posture and in nine when they were erect. The dose was one to four milligrammes per kilogram of body weight per day. Enteric-coated capsules were satisfactory. Weakness, drowsiness, nasal stuffiness and palpitations worried several patients, but severe toxic effects were rare.

Heparin and Coronary Insufficiency.

H. J. RUSSEK *et alii* (*J.A.M.A.*, July 12, 1952) discuss the effect of heparin in cases of coronary insufficiency. They state that it was suggested in 1949 that the effect of the anticoagulant drugs, heparin and dicoumarol, in cases of acute myocardial infarction and *angina pectoris* might be due to an increase in the coronary blood flow. In 1951, Graham and others reported considerable relief of symptoms in 55 out of 59 patients with *angina pectoris* who received weekly or bi-weekly injections of 50 or 100 milligrammes of heparin. The authors selected 14 patients suffering from *angina pectoris* with *S-T* or *T* wave changes. They were given 50 or 100 milligrammes of heparin at intervals of one to three days, and were tested by the Master two-step

test every twenty-four hours. After administration of 0·4 milligramme of glyceryl trinitrate these patients showed a decrease in *S-T* depression or *T* wave changes within five minutes. After heparin injections, however, no changes were observed in the electrocardiogram indicating increased exercise tolerance. The authors found that heparin injections did not increase the exercise tolerance and induced no signs of improvement in the electrocardiogram.

Cortisone and Renal Œdema.

N. M. KEITH *et alii* (*Arch. Int. Med.*, May, 1952) discuss the treatment of renal œdema with cortisone. They state that six patients were studied, and the results were very variable. Proteinuria increased in some and decreased in others. Sodium and water retention were noted when cortisone was given. In some cases the excretion of non-protein nitrogen, potassium and phosphorus was increased after cortisone administration. Calcium excretion was sometimes increased. In some cases, œdema vanished after administration of cortisone, in others no change occurred. The combination of cortisone with diuretics was advised when patients did not respond to treatment.

NEUROLOGY AND PSYCHIATRY.

Clinical Aspects of Cortical Behaviour.

MARGARET REINHOLD (*Brain*, December, 1951) describes a series of cases of disordered cortical function resulting from organic disease of the areas of cortex posterior to the Rolandic fissure. She puts forward a hypothesis that the dysfunctions described (dysphasia, dyslexia, dysgraphia *et cetera*) are dependent upon disordered aspects of perception, and that the nature of the dysfunctions may be expressed in terms related to perception. (Perception is defined as a phenomenon involving a series of events which may be scientifically described, but being itself a qualitative experience of an individual.) The dysfunctions may be regarded as belonging to several categories, which in themselves represent increasingly complex and integrated "levels" of cortical function; for example, there may be in turn imperfect immediate perception, defective image-formation of these percepts, defective images of symbolic representations of percepts, defective images of symbol abstractions, and defective power of abstraction, integration and association of perceptions and symbols. The author concludes that "mental" functions depend upon the ability to receive and abstract sensory stimuli of many different types and thence to proceed to compare, deduce and think in categorical terms. There is thus a relation between mental and physical phenomena which depends on the dynamic activity of neural tissue. It seems that no attempt should be made to "localize" any single function to any specific area of neural tissue in the post-rolandic area of the cortex. There does not appear to be a certain organization of function in the cortex. However, it is also known that one hemisphere may "take over" the functions attributed to the opposite hemisphere, so that some cortical cells are capable of multiple functions. It is also known that the later the evolutional development of cells, the more highly involved are their functional patterns. The brain is hence regarded as a plastic structure, and the author considers that perceptions dependent on the brain function are equally supple. She therefore puts forward a hypothesis that the cortical functions discussed depend upon perception and suggests that each dysfunction may be related to a particular aspect of disordered perception. She further suggests that each sensory stimulus may generate a specific rhythm, transmitted by the neural cells. These rhythms may vary from the crudest (in the newborn, for example) to the more complex, which may be abstracted and condensed and given symbolic representations by the cortex. It is not suggested that these rhythmic patterns which precede perception are necessarily electrical in nature.

Thalamic Activity in Stupor.

DENNIS WILLIAMS AND GERALD PARSONS-SMITH (*Brain*, December, 1951) report two cases in which there were clinical signs of a lesion in the mesencephalon. Electroencephalographic records were made on the patients when they were in a state of akinesis and mute, and also on recovery. The authors state that spindles of electrical rhythms were recorded from the postero-lateral region of the thalamus during stupor only, and that these spindles, which have not been recorded from the human thalamus in other conditions (including coma), were associated with episodic waves in the cerebral cortex. These episodic waves were abolished by "Pentothal" anæsthesia, but this made the spindles even more frequent and their rhythmic character more evident. The authors state that findings such as these have been made by other workers in the thalamus and cortex of cats in which the reticular substance of the mesencephalon has been divided. The present observations on the two patients have been related to the result of animal experiments and to the present knowledge of thalamo-cortical mechanisms. The authors suggest on the basis of the report that in akinesis with mutism (or stupor) the inactivity results from disturbances in the ascending reticular system, which is concerned in the integration and activation of afferent impulses.

Prognostic Factors in Insulin Therapy.

MAX COHEN (*Arch. Neurol. & Psychiat.*, October, 1951) states that he has studied 1000 patients, 639 by statistical analysis, in an attempt to discover prognostic criteria in insulin therapy. Factors apparently of no importance in the prognosis were race, marital status, religion, previous course of insulin shock therapy, number of treatment days, number of coma days, average daily dose of insulin, number of insulin convulsions, somatotype, diagnosis and age in relation to onset of the psychosis. The response to insulin therapy decreases with age, probably because of the increased length of time the patient has been ill. Patients who gain over 30 pounds in weight will do approximately twice as well as those who gain under 30 pounds. Patients who are ill for less than one year will do approximately twice as well as those who are ill for over one year. The author suggests that insulin therapy be given as early as possible in the first year of the illness.

Surgery and Disseminated Sclerosis.

SAMUEL J. ROSNER (*J. Nerv. & Ment. Dis.*, December, 1951) describes nine cases in which there were signs and symptoms which approximated to those of a multiple sclerosis syndrome. Myelography showed an obstruction in the high cervical cord region. In a series of 14 cases, a lesion was shown in 10 cases and confirmed by surgery in nine. The author considers that the clinical picture is due to restriction of blood supply by tumour or adhesions. He stresses the difficulty of the myelographic procedures, which call for special skill and great care.

Cysts of the Nerve Roots.

I. M. TARLOV (*Arch. Neurol. & Psychiat.*, July, 1952) reports his discovery in 1938 of sacral nerve root cysts and his establishment of their clinical importance as one cause of the sciatic syndrome. He states that these cysts are relatively common and may produce symptoms. They may result from a subarachnoid hæmorrhage infiltrating the nerve roots with absorption and cyst formation, or from ischæmic cell degeneration in the nerve roots. They occur chiefly at the junction of the posterior nerve root and its ganglion, under the posterior sacral arch, an area rarely explored surgically. The presence of cysts should be suspected when operation in the sciatic syndrome fails to reveal a herniated disk, or when there are slowly progressive changes referable to sacral or coccygeal portions of the *cauda equina*. Surgical removal of the cysts produces relief of the sciatic syndrome.

The Psychological Response to Steroids.

HOWARD P. ROME AND FRANCIS J. BRACELAND (*Am. J. Psychiat.*, March, 1952) studied more than 100 patients who were treated with ACTH, cortisone, hydrocortisone and related steroid substances for a variety of physical and a few psychological diseases. They state that the most striking psychological response was noted in the sphere of affect. In approximately 30% of cases the mood was elevated; this varied from mild to hypomanic states, associated with restlessness and insomnia. Some 25% to 30% of patients had affective changes such as anxiety, phobias or depression, the particular selection depending to some extent upon the predominating characteristics of the patient. Some 10% developed gross psychotic reactions; in almost all cases these tended to subside within a few weeks after cessation of the treatment. The authors state that withdrawal of cortisone or ACTH is fraught with two rather formidable complications: one is a feeling of fatigue and exhaustion, presumably due to temporary suppression of adrenal-cortical function; the other is more of a reactive state, when the patient is relatively suddenly precipitated into a painful, disabling, fully developed illness, in contrast with the usually slow onset of the initial sickness with its associated mental adjustments.

Special Articles for the Clinician.

(CONTRIBUTED BY REQUEST.)

LIV.

MEDICAL CERTIFICATION.

WITH the growing complexity of organized communities and to meet the requirements of legislation, accurate medical certification is essential to ensure the correctness of vital statistics; to aid in the control of infectious diseases; to enable employed persons to establish their rights to compensation and sick leave; to protect the employer—the Government or the private entrepreneur—against false claims; to assist the course of justice; and to protect the community at large. Governments have seen fit to impose certain statutory obligations on medical practitioners in relation to certification, and there is, in addition, a considerable body of legislation which is workable only by the provision of medical certificates. In the first category are the acts of the various States relating to the registration of the causes of death and the notification of infectious diseases, and in the second the acts relating to the care of the insane and inebriates, the workers' compensation acts. the repatriation acts, and legislation and industrial awards governing conditions of employment and entitlement to sick leave. In addition, proper medical certification plays an important part in systems of life assurance and superannuation, in recruitment for the armed forces and in the working of medical and hospital benefit organizations.

General Principles of Certification.

The basic essentials of a medical certificate are accuracy and clarity; and as the doctor is also accepted as an "expert witness", it is permissible for opinions to be expressed.

Therefore, in general, certificates have three components—a statement of facts observed by the doctor, information communicated by others, and one or more expressions of opinion. Certificates should also identify the patient by name and address and be signed legibly by the doctor with the addition of his qualifications, address and date, and should not be antedated or postdated.

Common Errors in Certification.

One of the commonest mistakes made in the writing of a certificate is to state as "facts" information transmitted to the doctor by the patient or his relatives. For example, "I certify that John Doe is suffering from a fractured femur resulting from a fall from his horse . . ." may or may not be true. Unless the doctor saw John Doe fall from his horse he should state, "I certify that John Doe, of ....., is suffering from a fractured femur. He states that the injury resulted from a fall from his horse . . ."; and such a certificate may properly be completed with one or more expressions of opinion by the addition of such words as "In my opinion the nature of the injury is consistent with the stated cause, and I consider that John Doe will be unfit for work for a period of at least three months from this date".

Carelessness in the writing of certificates does not enhance the reputation of the medical profession, and care should be taken that "facts" really are facts. It is rather bewildering for an insurance company dealing with workers' compensation cases to receive a series of certificates in relation to the same injury giving different diagnoses, such as, on one occasion reference to a "fracture of the distal phalanx of the ring finger of the left hand", and on another to a "fracture of the distal phalanx of the middle finger of the right hand". The doctor too who light-heartedly attributes his patient's disability to an industrial process with which he is unfamiliar may later regret his impetuosity, as did the doctor who certified that a tool-grinder was suffering from the effects of occupational dust, it being shown subsequently that the tools were ground under an oil bath and the procedure was entirely dust free.

The deliberately false and fraudulent certificate is in a category of its own and will be referred to later, but many certificates written carelessly in relation to absence from work and claims for sick pay are found, on investigation, to be not in accord with the facts and may result in the levelling of a charge of false certification against the writer. The employer presented by his employee with a medical certificate written on Thursday, the first occasion on which the doctor attended the patient, attributing absence from work for the preceding three days to "influenza" is justifiably critical when he knows that the absentee was seen intoxicated at the races on Wednesday. Consequently, pressure from patients to write "certificates of accommodation" should be resisted; but an employee who has been legitimately absent on account of illness, but who has delayed consulting his medical attendant, may be supported in his claims by a certificate in the form: "I have this day attended John Doe, of ....., who is suffering from influenza. He states that his illness commenced on ..... In my opinion his present condition is consistent with that statement."

The Question of Disclosure.

It is frequently suggested that disclosure of the nature of a patient's illness in a certificate is a violation of professional secrecy and that a diagnosis should not be given. While that argument may in certain circumstances be justified, in general it is not. The legislatures by statute insist that disclosure be made in relation to the cause of death and the nature of notifiable infectious diseases, and in Queensland under the *Medical Acts*, practitioners may be deemed guilty of professional misconduct if they fail to advise the Director-General of Health of indications of attempted or completed crime or illegal operation, of wounds from cutting instruments not accidentally incurred, of bullet wounds, of partial strangulation, of asphyxiation, of trauma caused by heat not accidentally incurred, and of trauma caused by electricity, in patients under their care. Certificates written in relation to sick pay, sick leave, workers' compensation, claims for hospital benefits and related matters become, in most cases, the property of the patient, and he decides their final destination, or they are supplied to third parties with his knowledge and consent, and there is therefore no breach of professional confidence. On the other hand, it may be quite legitimate at the doctor's discretion, and in the interests of his patient, to describe a carcinoma of the stomach as "stomach trouble" and in certain circumstances to use a euphemism in some gynæcological or obstetrical cases.

The Unnecessary Certificate.

In recent years there has been a growing demand by government departments, employers and retailers for medical certificates which have little or no relation to the welfare of patients. Such unreasonable demands on the time of doctors should be countered by refusal to write such certificates.

Some Special Forms of Certificate.

The following statements are based on legislation of the State of Victoria, but comparable acts exist in some, or all, of the Australian States.

The "Medical Certificate Concerning Death".

The common form of death certificate in use in the Australian States has recently, in Victoria, been slightly amended in accordance with the request of the World Health Organization, and a medical practitioner "who has been in attendance during the deceased's last illness" is obliged to complete and return such a certificate to the registrar within forty-eight hours of the death of the deceased. Even if the death has been reported to the coroner (and all cases of death from violence, accident or unknown cause come within the coroner's jurisdiction) the certificate should as far as is possible be completed and forwarded to the registrar, but in such circumstances the right-hand portion of the certificate, "Notice of signing medical certificate concerning death", must not be completed. In the writing of such certificates the nomenclature of "The International List of Diseases, Injuries, and Causes of Death" should be used. No fee is chargeable for a death certificate.

Cremation Certificates.

Provided the cause of death is known and the case is not one for the coroner, a body may be cremated on completion of two certificates in the proper form—one signed by the practitioner who attended the patient in his last illness and the other by either an appointed "certifying medical practitioner" or a medical officer of health. In addition, the signatories must not be in business relationship (for example, partners) or have a pecuniary interest in the death of the person concerned, and they must both be registered under the *Medical Act* of the State in which the cremation is to be carried out.

Registration of Stillbirths.

The registration of stillbirths is compulsory in most of the Australian States, and when a medical practitioner has been in attendance he must certify, on the appropriate form, to what was, in his opinion, the cause of death.

Notification of Infectious Diseases.

It is the duty of a medical practitioner who becomes aware that any patient under his care is suffering from one of the diseases which have been declared as "notifiable", to inform the municipal authority of the area in which the patient resides of that fact.

Notification of Venereal Diseases.

Venereal diseases are notifiable to the chief health officer of the State, in the first instance by number; but if the patient defaults in his treatment and no intimation is received from another practitioner that the patient has been transferred to his care, the patient's name and address must be disclosed to the chief health officer, who is empowered to take further action to ensure that proper treatment is resumed.

Certification under the Lunacy Acts.

A person insane or apparently insane may be placed under care and restraint in one of the following ways:

1. By committal to an institution by a justice on the certificate of one medical practitioner or by the order of two justices without a medical certificate.

2. By admission to a "receiving house" on the certificate of two doctors (not standing in blood or professional relationship and not related to the patient) that the person concerned is "apparently insane". Such certificates are valid for seven days.

3. By admission to a mental hospital on the certificate of two doctors (not standing in blood or professional relationship and not related to the patient) that the person concerned is insane. Such certificates are valid for seven days.

4. By becoming a "voluntary boarder" without medical certification. The patient must be examined in the State in which he is certified and the writer(s) of the certificate(s) be registered in that State. As certification under the *Lunacy Acts* deprives a patient of his liberty, and as claims for substantial damages have been based on allegations of improper certification, the utmost care should be taken in certifying persons as "insane" or "apparently insane".

Certification under the Inebriates Act.

The requirements in regard to certification under the *Inebriates Act* are complex, and it is advisable for the relatives of the patient to enlist legal aid before seeking the one medical certificate required.

The Penalties for False and Careless Certification.

Penalties for false and careless certification are both general and specific; general to the profession, in that slipshod certification by a minority of practitioners has led to a general decline in the respect paid to the integrity and good faith of the medical profession, and specific to the individual in relation to the powers of the medical boards.

It has been stated by the medical officer to a semi-governmental organization in Victoria that twenty years ago he would be informed: "These claims are in order, they are supported by medical certificates; but will you examine these others to which no certificates are attached?" Today he is asked: "Will you look at these certificates and tell us if they are any good?" Other medical men employed in industry and in official posts repeatedly deplore the careless and, in many cases, nonsensical certificates with which they have to deal in the course of their duties.

The general standard of certification can be improved only by the efforts of the individual doctor; but if the individual continues to err he is at peril of being dealt with by the medical board under which he is registered. Many years ago the General Medical Council in Great Britain issued a "warning notice" that it would regard the giving of untrue, misleading or improper certificates, notifications or reports as coming within the definition of "infamous conduct in a professional respect", and the medical boards of the Australian States hold similar views.

C. H. DICKSON,
Melbourne.

British Medical Association News.

SCIENTIFIC.

A MEETING of the New South Wales Branch of the British Medical Association was held on October 25, 1952, at the Hotel Canobolas, Orange, DR. R. H. MACDONALD, the President, in the chair.

Peptic Ulcer and its Complications.

DR. S. J. M. GOULSTON read a paper entitled "The Treatment of Peptic Ulcer and its Complications" (see page 291).

DR. F. F. RUNDLE read a paper entitled "Surgical Aspects of the Treatment of Peptic Ulcer" (see page 293).

DR. J. T. PATON said that he had been interested to note how akin were the ideas of the physician and the surgeon who had spoken, about the diagnosis and methods of treatment. Treatment from the medical side did not seem to have altered since he had been in practice, but the attitude of the practitioner towards possible surgical intervention had improved. Most practitioners were anxious to see that nothing was missed and that surgical intervention was called for in time.

DR. K. C. T. RAWLE also remarked on the similarity of the views expressed by the physician and the surgeon. Sir Arthur Hurst had once said that if he had the opportunity of putting a patient to bed for three weeks, he could do almost the same wonders as the surgeon could do. The economic problems presented by patients with ulcers were considerable; a man who was out of work found it hard to carry on with medical treatment. The financial position had to be considered. Dr. Rawle said that he wished to stress the necessity for vitamin *C* for the healing of ulcers. In Orange, though patients might be short of vitamin *C* when they started their treatment, they were short of vitamin *B* when they finished treatment; they presented with bleeding lips, keloids *et cetera*. A year or two of ulcer diet in Orange led to such a deficiency; it was characteristic of that particular area.

DR. L. W. TUNLEY quoted the case of a patient whose improvement after gastrectomy did not support Sir Arthur Hurst's remark, quoted by Dr. Rawle. The patient was a motor-lorry driver, in the fifties, who worked very hard and did heavy lifting; he had one or two massive hæmatemeses. He was kept in bed two or three times for a month at a time in hospital and treated with rest, diet and medical measures; he was a most cooperative patient. After the second spell of about six weeks, he was sent to Dr. Rundle for gastrectomy. Dr. Tunley said that he had seen the patient several times in the twelve or eighteen months that had elapsed since the operation, but not professionally. On the last occasion, about one month prior to the meeting, the patient, with two other men, was carrying a heavy refrigerator which he delivered to Dr. Tunley. The patient said that he had never felt better in his life, he could eat anything, and had three large meals a day, and had had no trouble since the operation. Dr. Tunley said that he could endorse all that Dr. Rundle had said about the surgical aspect of treatment.

DR. J. H. PRIESTLEY said that he had been interested to hear Dr. Rundle advocate the Hoffmeister-Polyá type of gastrectomy; one school in England was going back to the Billroth I operation. He asked whether Dr. Rundle could give the indications for the latter. He also asked Dr. Rundle to comment on the "dumping" syndrome that sometimes occurred after gastrectomy.

DR. H. R. HODGKINSON referred to the numbers of medical practitioners he knew who had gastric and duodenal ulcers and who had not undergone gastrectomy. He said that if one removed four-fifths of the stomach, leaving a small remnant that would hold a pint with an outlet the size of a florin, one not only removed the stomach, but also impaired pancreatic and biliary function. If a short-circuiting procedure as directed by Moynihan was carried out from the lesser to the greater curvature behind the transverse colon, the stomach was decompressed and deflated, and a great number of ulcers would heal. Dr. Hodgkinson quoted three cases, one of a shearer, who had an enormous gastric ulcer eroding the pancreas, and nearly bled to death, and two of patients with severe duodenal ulcers; all three patients were restored in weight and working capacity by the Moynihan short-circuiting operation. At operation on the man with the enormous gastric ulcer thick chromicized gut had been put in, and he bled no more. Now one could use gelatin sponge, hæmoplastin and vitamin *K* to help. Dr. Hodgkinson asked why, if gastric resection was so wonderful, so few doctors had it. He also wondered why Moynihan's

old short-circuiting operation was so much abused, when a man subjected to it could make up weight and be bright and happy, whereas after gastric resection patients were always apt to develop pernicious anæmia. Dr. Hodgkinson felt that even if an ulcer was malignant and resection was performed, operations for gastric malignant disease were always bad. Death occurred within four or five years. If there were secondary deposits in large numbers around the head of the pancreas, they could never be removed.

DR. J. E. F. DEAKIN said that the speakers had not stressed the medical treatment of perforation. For years he had carried out the ordinary standard suture of every perforated ulcer. In February, 1949, a female patient, aged twenty-six years, had eaten sausages for breakfast, and her ulcer perforated about two hours later. Dr. Deakin, called in consultation, saw the patient about eight hours after perforation had occurred, with a view to operating on her. She had been given morphine about 2 p.m. and her condition did not seem very bad. He passed a Ryle's tube, and gave her intravenous saline and dextrose therapy for three days, then the usual ulcer treatment by mouth. She made an uninterrupted recovery. Another woman was examined about five or six hours after perforation had occurred. She was also treated medically and recovered. A third patient, going home from work, had drunk several beers, and his ulcer perforated during the night. The next morning examination disclosed typical symptoms of perforation, rigidity, tenderness *et cetera*. His condition was not particularly bad. When the patient was seen about twelve hours after the perforation, Dr. Deakin told his resident medical officer to pass a Ryle's tube; he said that he would re-examine the patient later and probably would not operate. The resident was obviously uneasy. About an hour and a half later Dr. Deakin still refrained from operating and the man made a perfect recovery. Another man whose ulcer had perforated twelve hours before admission to hospital had responded to conservative treatment. X-ray examination revealed gas beneath the diaphragm. The last-mentioned case had occurred ten days before the time of the meeting. Dr. Deakin said that he still would not contend that the treatment described in his cases was ideal; surgery was indicated in many cases. He thought that medical treatment was practically the same as surgical treatment had become. Years earlier the custom had been to suture the ulcer, flush out the peritoneal cavity with saline, and put in three drains. Surgical treatment nowadays was mostly suture without drainage. Now he aspirated the gastric contents, gave intravenous saline and dextrose therapy for three to four days, and then gave the usual ulcer diet by mouth. In most cases in which operation was performed, something, most frequently a piece of omentum, was found adherent at the perforation. One put in a purse-string suture, if possible, and if the ulcer was big and hard and insertion of a purse-string suture was impossible, the suture had to be put right through the ulcer wall and the omentum sutured to it. He wondered what the difference was between doing that and aspirating the stomach and letting the ulcer heal. Dr. Deakin thought that if the patient was not examined until six to twelve hours after the perforation had occurred, and if tenderness and rigidity did not extend right down to the pelvis, medical treatment should be given. If great shock was present, and tenderness and rigidity were present all over the abdomen and those conditions still persisted after a dose of morphine, operation should be performed. In almost all cases of perforation, morphine should be given straight away, but not if a consultant was to be called in, because it might give the consultant a different impression from that of the medical practitioner in attendance. Referring to partial gastrectomy, Dr. Deakin said that if symptoms recurred three times after medical treatment, operation should be performed. If it was discovered that there were large adhesions or much inflammation round the duodenum, one should not go right down and excise the ulcer, but leave about half an inch or more and invert it. The duodenum should be closed in two or three layers; if chromicized gut was used, care should be taken to see that it was thick enough. If it was too thin, early absorption of the gut and leakage might occur.

DR. A. J. COLLINS said that it was an important observation that a surgeon and a physician, each an expert in his branch of the work, had reached comparative agreement on all points with regard to treatment and the indications for surgery. It therefore looked as if the advent of the newer knowledge, aided by an increased study of the pathology of ulcer, increased diagnostic methods and improved statistical methods, was making the present knowledge of the subject fairly complete. Both speakers had emphasized the fact that a study of each individual case was important, and that a knowledge of the life history of ulcer must be applied to the patient under treatment, so that technical knowledge was tempered with and balanced against psychological knowledge of the patient. Both speakers had laid before the meeting as nearly complete a picture of medical and surgical treatment as could be wished for. Dr. Hodgkinson had emphasized the confusion existing in regard to individual cases. In the good old days certainly many patients treated by simple gastro-enterostomy recovered. But no doubt they would have recovered with medical treatment. Perhaps it was not right at that late stage to attribute their recovery to the type of surgical treatment given. A large number of patients from different centres must be considered. Dr. Collins said that he saw Dr. Hodgkinson's point of view, but he was on the side of the men who had read the papers. He thought the large operations mentioned were justified and got good results. Dr. Collins asked Dr. Goulston to say something about the object of effective neutralization of gastric acid. He said that his (Dr. Collins's) namesake in America some years earlier had said that it was to reduce the pH to a figure lower than 3·0; digestion of the gastric wall could not take place if it was higher. In hospital practice he always considered neutralization was effective when the patient's pain ceased and he began to feel well. So long as the pain persisted, it was probable that neutralization of gastric juice was not effective. Dr. Collins's second question concerned the use of "Amphotabs" and similar substances, which were placed in the mouth and sucked all day long. He asked whether they were as effective as neutralizing agents given by the intragastric drip method. Dr. Collins's third question related to hæmorrhage; he asked for some comment on renal insufficiency occurring in association with hæmorrhage. The suggestion had been made previously that blood urea estimations should be made at the bedside to determine how dangerous the hæmorrhage might be. Referring to the danger of hæmorrhage to a patient aged over forty years, Dr. Collins said that he had found surgeons very timid in agreeing to operate. One patient of his had died after four surgeons had held numerous consultations about him; good surgeons would not operate on him. That patient had had enormous hæmorrhages every few weeks. Dr. Collins said that practitioners passed through many phases in the treatment of the diseases under discussion, and when the Sippe treatment was introduced in 1915 they thought that they had the answer to the whole problem. But although the results of that treatment were infinitely better, they still found patients with huge ulcers requiring surgical treatment. Dr. Collins wondered whether the condition was on the increase, and whether the early and effective treatment of early and uncomplicated ulcer was lessening the incidence. It might be that the early ulcers had smouldered on with little or no clinical symptoms, or that a higher proportion of uncomplicated ulcers became chronic. In view of the increased skill of surgeons, the increased efficiency of their team of assistants, better anæsthetics, and the availability of blood transfusions, they would have to reconsider the whole question of peptic ulcer. Dr. Collins thought that they would have to review their whole attitude to it, since surgery was no longer the great risk that it used to be.

DR. J. RUTHERFORD asked Dr. Rundle in what form the 10% of failures mentioned were manifested, and what was to be done with them.

Dr. Goulston, in reply to Dr. Collins's question about the effective neutralization of gastric juice, said that he did not know a quantitative method that would be useful to the general practitioner or the physician to indicate the amount of effective neutralization by antacid or neutralizing substances. The best indication was the relief of pain. Patients differed in the relief obtained from alkalis, and what suited one might be of limited value to another. The exhibition of atropine or something having the same effect was very important, and full dosage should be used. Most of the modern antacids, including "Amphotabs", were useful, but the most important neutralizer was food of any type, particularly milk. In reply to Dr. Collins's question about renal insufficiency, Dr. Goulston said that estimation of the blood urea level was very helpful; it should be made as a routine procedure. The level might be 100 milligrammes *per centum* after hæmorrhage. If it reached 180 milligrammes *per centum* it was an indication of alkalosis, either temporary or due to some renal defect. On the question of operation, Dr. Goulston said that those who had spoken showed that certain patients did not do well after it. Dr. Hodgkinson's remarks were important. Dr. Goulston felt strongly that in uncomplicated duodenal ulceration surgical intervention should not be recommended. He felt suspicious of surgical statistics, especially those from the United States, as often one did not know what were the indications for operation. When the indication was sound, surgery in good

hands was effective, and the cases in which such treatment went wrong were few. Statistically one could not overlook partial gastrectomy as the operation of choice. Dr. Goulston said that he did not like any other unless as an emergency measure.

Dr. Rundle, in reply to Dr. Priestley's question about the Billroth I operation, said that he did not want to suggest that only one operation was any good. A point in favour of the Billroth I procedure was that it was physiological, food was held in the stomach and fed on into the duodenum in natural fashion. Dr. Rundle said that he had watched the late Professor Grey Turner perform the operation. He was one of its advocates in England. A sound policy with resections like that for peptic ulcer was to learn a standard technique and have it as well practised and as nearly fool-proof as possible. His own training had been in the operation described. There were objections and difficulties in performing an occasional Billroth I operation. There might be tension on and leakage at the anastomosis. Another was the difficulty of resecting an adequate length of stomach. Sometimes a patient had a double ulcer with some fixation of the stomach. But no doubt the operation was giving very good results in the hands of people performing it frequently. Referring to the "dumping" syndrome, and the fact that doctors were said not to undergo operation for ulcer, Dr. Rundle said that he had sought out in the last few months five doctors and questioned them about their gastrectomies. All were very strong advocates of gastrectomy, and all had told him that they wished they had had the operation performed months or years earlier than they had had it. One of the doctors had been rather bothered by the "dumping" symptoms, including epigastric pain, sweating, a feeling of tightness and malaise after meals. Those manifestations had gradually grown less and less, and he was now free from them. If the patient would take more frequent feeds and lie down for twenty minutes after meals during the post-operative period, the problem would often solve itself. On the subject of pernicious anæmia, Dr. Rundle said that the question had been examined, and it had not been established that it was more common in patients who had undergone gastrectomy than in the general population. Referring to the patient with hæmorrhage mentioned by Dr. Collins, Dr. Rundle said that he thought it was becoming clear that a surgeon's job was to operate after rapid resuscitation in the operating theatre with his team all ready. The patients requiring surgical intervention were precisely those that could not be maintained in the ward with blood transfusion. Therefore blood transfusion was only of resuscitatory value, and the place for it was in the operating theatre, the source of hæmorrhage being controlled as soon as its effect was maximal. When it was carried out expectantly in the ward, its effect was disappointing and that led to procrastination and fatal delay. On the question of patients with large chronic gastric and duodenal ulcers appearing in great numbers, Dr. Rundle said that that might depend on the fact that peptic ulceration as a whole was becoming more common; its incidence was growing all the time. Dr. Rutherford's question about failures raised a very important point. Dr. Rundle did not see how it was possible to compare results from clinic to clinic unless there was agreement first of all on the indications. It was better for everyone to study his patients carefully beforehand. The more nearly perfect the indications were for surgery, obviously the better the results would be. Dr. Rundle did not know what to do for patients with a bad result after gastrectomy who had primarily a mental upset or developed one after operation. The best treatment was prophylaxis. Surgeons should not operate on patients unless they were sure the symptoms were due to ulcer and not to some associated condition or mental disorder.

Generalized Lymphadenopathy.

DR. A. E. MCGUINNESS read a paper entitled "The Significance of Lymphadenopathy" (see page 285).

DR. V. J. MCGOVERN read a paper entitled "The Significance of Generalized Lymph-Node Enlargement" (see page 288).

DR. S. R. DAWES said that the speakers had given a full analysis of the subject. There were some points that could be dealt with. It seemed at the present time that attempts to diagnose accurately one out of the group of conditions discussed were rather like hair-splitting and were perhaps not justified. The patients' progress varied; they could die quickly, or they might last for years. Examination of sections from different parts of the same patient showed different processes at work at the one time. Dr. Dawes thought that clinicians could restrict the name and call the condition a reticulosis following a pattern; that would be better than trying to split it up into classes. He had been asked in cases of leuchæmia in which primitive cells were present to say whether it was myeloid or lymphoid; he did not think it possible in all cases to do so. He had given an opinion sometimes, and found his diagnosis upset some months later. When a cell was very primitive, it was impossible to say what it was. It might be an abnormal cell of a series. Referring to the question of the incidence of the reticuloses as he saw them, Dr. Dawes said that many practitioners, as soon as they examined a patient so affected, sent him at once to the city for diagnosis; he therefore could not assess the incidence. Thanks to their reading of a popular magazine, people were now awake to diseases like leuchæmia, and rushed a child to their family doctor at once on suspicion, wanting to know whether he had leuchæmia or not. At the present time most cases of lymphadenopathy were acute and had a duration of a few weeks. In some cases with a short history, after a few weeks had elapsed the enlarged glands would be found to disappear and there was no need for further worry. Referring to generalized adenopathy, Dr. Dawes said that even in that disease in which the glands were generally enlarged, it did not follow that it was so in all patients seen; it might be so only in the neck or axilla, and the process might extend after a time. Dr. Dawes said that Hodgkin's disease was rare in the district. With regard to the acute cases, which were relatively common, the majority of patients did not have glandular fever. The condition might last for some weeks, and the neck and armpits might be affected. The other most common of the conditions was the lymphatic type of leuchæmia. Leuchæmia in children was a more common disease; they were able to diagnose it at the present time without a very great increase in the leucocytes. Cases of generalized glandular enlargement were not very numerous, and they did not see the condition as it figured in the text-books of some years before. That was probably due to the better publicity that had been given to it. Dr. Dawes said that a case could be made out for regarding the conditions under discussion as probably phases of one process. One sure thing was that on clinical grounds they were invariably fatal, much more so than the average malignant growth. He had been interested in the remarks about folic acid antagonists. A remission that seemed remarkable had been induced in one case; all other cases in his experience had terminated fatally.

DR. H. BUSBY gave briefly the history of the patient mentioned by Dr. Dawes. He was a boy, aged ten years, in whom leuchæmia had developed just under a year previously. He was sent to Professor Lorimer Dods, at the Institute of Child Health. Dr. Busby understood that at that time Professor Dods had had a communication from Dr. Diamond, of Boston, describing treatment with cortisone and aminopterin. The boy was given a course of aminopterin and that was still continuing. Cortisone had not been given. At the time of the meeting he was fairly well. He had occasional nose bleeds and some ulceration in the mouth and throat. Dr. Busby suggested that Dr. Dawes should say more about his blood picture.

Dr. Dawes spoke again. He said that the child had not had at first a high total leucocyte count; the leucocytes numbered 20,000 per cubic millimetre at the start, over 90% being immature mononuclear cells. No abnormal cells had been seen since the child had returned home. At the time of the meeting his hæmoglobin value was well over 100%. An attack of measles had had no effect on the leuchæmia. Platelets were present in normal numbers. The treatment had produced complete baldness, even to the loss of the eyebrows.

DR. JAMES ISBISTER observed that there had been only partial agreement in their papers between Dr. McGuinness and Dr. McGovern; but they seemed to disagree a good deal on one basic point, and that was the question whether the diseases under discussion were all one condition or not. Dr. Isbister believed that many ran an unpredictable course; two biopsies might be carried out and the reports might be different. His clinical experience had been that on the whole biopsies were not very helpful in relation to the probable course. It seemed that there was a difference of opinion between pathologists. He knew pathologists who said that it was difficult to carry out a lymph gland examination and know definitely what they were looking at. Dr. Isbister asked Dr. McGovern to say whether it was possible always to distinguish between the reticuloses and other conditions such as mononucleosis, brucellosis and so on. He also asked what line of investigation was likely to be most useful in the generalized lymphadenopathies. Those most used were examinations of the peripheral blood, the sternal marrow and lymph node biopsy.

Dr. McGuinness said that Dr. McGovern's paper had been notably anatomical, since his work was concerned with the terminal phase of the diseases under discussion. That was

important, in so far as it bore out the contention that a biopsy was vital; if the patient had a reticulosis, the prognosis was grave. The clinical pattern of the disease, especially during the first month, would give some idea of the prognosis. It was impossible to be absolutely certain, because at all times the conditions were unpredictable. Dr. McGovern had mentioned surgery and irradiation as forms of treatment, and had quoted a case in which radical surgical treatment many years previously had resulted in cure. Dr. McGuinness said that on the other hand countless cases were mentioned in the literature in which the survival period after irradiation had been just as long. He did not think surgery was vital, but that was a line of investigation that should be followed. The result probably depended basically on the nature of the disease process itself, and it seemed to be due to good fortune that the patient mentioned by Dr. McGovern had been treated surgically. Dr. McGuinness stressed the need for carrying out a biopsy because the diagnosis was important to the patient and to the doctor.

Dr. McGovern, in reply to Dr. Isbister, said that at the Royal Prince Alfred Hospital he saw many lymph nodes from patients with generalized enlargement in which no lethal disease could be diagnosed. The condition was a reactive hyperplasia—the patient was reacting to some stimulus in a normal physiological way. That was all one could say. But if a lymph node was involved by one of the diseases under discussion, one could usually diagnose it. Of course not all lymph nodes were affected. Sometimes a lymph node was removed which had been wholly converted to fibrous tissue. No diagnosis could then be made. There was no really serious difficulty in differentiating between Hodgkin's disease and follicular lymphoblastoma. With reference to Dr. McGuinness's comments on irradiation, Dr. McGovern said that the lymphocytes were the most sensitive cells to irradiation in the body, and the various derivatives of the primitive cells from which they were derived were very sensitive. But the primitive reticulum cell itself was not very sensitive to irradiation. Consequently in the diseases under discussion sooner or later after irradiation there would be recrudescences. Dr. McGovern said that he had known other patients alive and apparently well for a number of years; those were cases in which attention had been called to the disease at an early stage, when it was localized, for example, in the alimentary tract. One patient had been operated on three years earlier, and was still healthy. She had had a volvulus, and at operation the surgeon could see nothing wrong with the involved portion of the gut. He excised it, feeling that something was wrong, and pathological examination revealed lymphosarcoma involving a section of the bowel about eight inches long. Dr. McGovern said that for him the diseases were clear cut. He saw them in the early and in the late stages, and they followed a certain course.

Conclusion.

Dr. Macdonald, from the chair, thanked Dr. Goulston, Dr. Rundle, Dr. McGuinness and Dr. McGovern for their papers, and for the careful thought and industry that had been expended in their preparation. He also thanked those who had joined in the discussion and added to the interest of the meeting. He said that the fact that some 55 members of the Association had attended the meeting, and that some had come from distant centres, must be a great source of satisfaction to those who had presented papers, and must repay them for the effort and time which they had spent. The success of the meeting supported the policy of the New South Wales Branch Council in its decision to conduct one meeting each year in a country centre. Dr. Macdonald asked Dr. Dawes to convey the thanks of the Council to the President and members of the Western District Medical Association for all that had been done to make the meeting possible.

A MEETING of the Victorian Branch of the British Medical Association was held on June 18, 1952, at Saint Vincent's Hospital, Melbourne. The meeting took the form of a number of clinical demonstrations by the members of the honorary medical and surgical staff of the hospital. Part of this report appeared in the issue of February 21, 1953.

An Unusual Case of Carcinoma of the Lung.

DR. F. COLAHAN'S first patient discussed was a male, aged sixty-one years, who had presented with a two years' history of repeated hæmoptysis, right axillary pain (a dull ache, increasing over the past eight months), a lump, small and hard, on the right medial axillary wall, and the loss of three stone in weight in three months.

On examination of the patient, he appeared a sick-looking old man, rather cyanosed. The trachea was deviated slightly to the right, and there was considerable clubbing of the fingers and toes. Small, moderately hard, tender glands were present in both axillæ. On examination of the chest, the left hemithorax moved better than the right. From the anterior aspect, vocal fremitus and vocal resonance were absent in the right mid-zone and the percussion note was dull. From the posterior aspect, sonorous expiratory rhonchi were present, and whistling râles were heard at the bases of both lungs. The Casoni test produced a negative result.

The patient had had every conceivable radiological investigation performed in a country centre. The upshot of these was the discovery of a solid, rounded opacity in the right mid-zone. This was approximately one and a half inches in diameter. The following pathological report was made after biopsy of a gland from the right axilla: "Clear cells uniform in size . . . with abundant cytoplasm and a large nucleus. . . . The appearance suggests an origin from a primary renal carcinoma."

In the light of these observations, the provisional diagnosis of carcinoma of the kidney with a "cannon-ball" secondary deposit in the lung was made. A second excretion pyelogram revealed a slight deformity of one calyx of the right kidney not noted in the earlier excretion pyelogram. Suddenly, the patient developed a violent fit of productive coughing. A further X-ray examination revealed that the solid tumour of the lung had now cavitated and presented a fluid level. This threw some doubt on the provisional diagnosis.

The patient's condition grew worse, as did his pain, and his cough became more troublesome. He expressed the desire to go home and thus left hospital. He died about two weeks later. A post-mortem examination performed in the country revealed a primary carcinoma of the lung with secondary spread to the axillary glands and left suprarenal. The kidneys were normal.

Gastro-Jejuno-Colic Fistula.

Dr. Colahan then showed a male patient, aged forty-nine years, who had had a posterior gastro-jejunostomy performed in 1926 for an anterior duodenal ulcer. From 1926 to 1938 he had suffered from mild infrequent attacks of indigestion. In 1940 he had been admitted to hospital with a severe hæmatemesis requiring blood transfusion, but after this he remained free of any symptoms until three months prior to his admission to hospital, when he complained of the following: (i) diarrhœa with the passage of five to twenty yellow fluid stools per day; the onset was fairly sudden; (ii) vomiting, after a bowel motion, of yellow fluid with a definite fæcal odour and taste; the vomiting was also of sudden onset; (iii) flatulence—copious belching of gases that smelt like flatus; (iv) pronounced weight loss—two stone in three months. Nausea and anorexia were also present, and the patient was greatly distressed because of his abhorrence (to use his own words) of his bowels acting through his mouth.

On examination of the patient, no abnormality was detected. Yellow formed stools were being passed. Investigation of the blood chemistry revealed some lowering of the serum protein content. A simple test by the use of ingested passion-fruit was carried out. Seeds appeared in a bowel motion three hours after ingestion, but this result was deemed inconclusive. A barium enema flowed freely from the transverse colon to the stomach, establishing the presence of a fistula. The patient vomited formed fæces during the enema.

At operation the transverse colon was found to be adherent to the site of the gastro-jejunal anastomosis, and a fistulous communication admitted one finger. The fistula was unpicked, the colon and jejunum were repaired and a subtotal gastrectomy was performed. The patient was given three pints of blood during and just after operation. His convalescence was uneventful, and he left hospital feeling very well, with a good appetite, on his eighteenth postoperative day.

A Case of Diabetes Insipidus following Severe Head Injury.

The third patient shown by Dr. Colahan was a young man, aged eighteen years, who had been brought to the casualty department after a motor bicycle accident. His injuries at this time were the following: head injuries, cerebral irritation, fracture of the anterior cranial fossa, partial avulsion of the scalp, fractured nasal bones, fracture of the mandible

and fracture of the left clavicle. He was unconscious for eight days, and on his recovery it was noticed that his left arm was weak. Examination revealed a branchial plexus lesion, apparently an avulsion injury—a lesion of the upper and middle portions of the plexus; the upper part very severely involved, the middle partially. He was treated on an abduction splint and with physiotherapy, and improvement was occurring.

After the patient's discharge from hospital he noticed several abnormalities. He had intractable thirst—he would drink "a kerosene tin full of water" daily. He also had polyuria and dryness of the skin. He was readmitted to hospital with the diagnosis of *diabetes insipidus* and treated with 0·5 millilitre of pitressin every eight hours. This reduced his fluid intake from 250 to 90 ounces per day and the output from 320 to 90 ounces per day. The specific gravity of his urine on his admission to hospital was 1000, and on his discharge 1007. At the time of the meeting he received five units of pitressin tannate in oil every forty-eight hours, and was symptom-free.

Neurilemmoma of Superior Thoracic Sulcus.

The next patient shown by Dr. Colahan was a male, aged sixty years, who had suffered from a "blackout", and radiographic examination of his chest revealed a circular mass in the thoracic inlet, situated in the posterior mediastinum, giving rise to no symptoms. The tumour was removed and found to be a neurilemmoma on differential staining by van Gieson's method. It was a rounded mass about two inches in diameter, and apparently arose from an intercostal nerve. The patient's convalescence was uneventful.

Diaphyseal Aclasis of the Scapula.

Dr. Colahan then showed a male patient, a wharf labourer, aged twenty-five years, who had noted increasing difficulty with lifting associated with his work. X-ray examination revealed a partially calcified tumour growing from the ventral surface of the scapula and displacing it posteriorly. It was excised with partial scapulectomy, and the patient resumed his occupation. Before section the tumour appeared to be a chondroma with typical cartilaginous hummocks. But when sections were prepared it could be seen that the hummocks were merely cartilaginous caps on normal bone. There were one or two patches of necrotic bone in the mass, which was classified by the pathologist as diaphyseal aclasis (Keith).

Osteogenic Sarcoma of the Femur.

Dr. Colahan's next patient was a young man, aged eighteen years, who had a nine months' history of a swollen left knee. The swelling had followed minor trauma, and for about one month was treated as subacute osteomyelitis. X-ray examination revealed an osteogenic sarcoma of the lower end of the femur.

On his admission to the hospital he had three months' deep X-ray therapy, resulting in some recalcification of the growth as was shown in serial X-ray films. However, pain was now his leading symptom, and it grew worse daily. Tremendous sedation was required for its relief.

It was decided to perform a disarticulation through the hip solely for the relief of pain. The operation was successful in that regard, and some enlarged femoral and iliac glands removed at operation were free of tumour. In his first three monthly check-up the patient's chest was radiologically clear, and he was well.

Adenoma of the Bronchus.

Another patient shown by Dr. Colahan was a married woman, aged fifty-one years, who had been admitted to a medical ward six months prior to operation, with the diagnosis of unresolved pneumonia. When that had cleared an opaque area was visible in the X-ray film, situated in the left mid-zone, and measuring about one and a half inches across. It was a rounded area with a somewhat furry edge, suggestive of hydatid disease. However, both the Casoni test and the hydatid complement-fixation test produced negative results.

She was readmitted to hospital with a history of fatigue, repeated small hæmoptyses, cough and sputum, and chronic upper respiratory tract infection. The opaque area had increased in diameter to about two inches. Bronchoscopic examination gave negative results.

Operation was undertaken to remove what was thought to be a hydatid cyst. The mass was being shelled out of a false capsule quite readily when sudden profuse bleeding occurred from the lung root, necessitating tourniquet lower lobectomy. The pathological report was as follows: "The tumor is growing in the lamina propria of the bronchial mucosa, spreading outwards and surrounding bronchial cartilage, and growing into surrounding lung. There is a condensation of fibrous tissue between normal lung and tumor. The tumor is formed of clear, uniform, cuboidal cells with round nuclei arranged in solid tubules. In some places the tubules have acquired a lumen. The appearances are typical of the so-called Adenoma of the Bronchus."

Varicose Ulcers Treated by Nylon Mesh Stockings.

Dr. Colahan finally showed two patients with varicose ulceration, in whose treatment nylon mesh stockings were used as a means of elastic support. The results were very encouraging.

The Kimmelstiel-Wilson Syndrome in Diabetes Mellitus.

DR. W. HAMILTON SMITH presented cases and photographs of pathological material illustrating the feaures of the Kimmelstiel-Wilson syndrome in *diabetes mellitus*. The clinical features which characterized the syndrome were albuminuria, hypertension, œdema, diabetic retinopathy and neuropathy. The pathological basis of the syndrome was renal intercapillary glomerulosclerosis associated with micro-aneurysms of the retinal capillaries.

Sarcoma of the Skull.

DR. FRANK MORGAN showed a female patient, a widow, aged sixty-seven years, who gave a history of having struck the right side of her head on a household fitting about twelve times in the last nine years. Two years prior to the meeting a painless swelling had appeared at that site. It had commenced to grow rapidly only in the last two months. There were no other symptoms, and general examination gave negative results. The lump measured three inches in diameter and projected one and a half inches above the surface of the skull in the right fronto-parietal region. It was firm and attached to bone and to the overlying skin, and vascularity was considerably increased. There were no other abnormal clinical signs; the cerebro-spinal fluid was normal in all respects and the blood failed to react to the Wassermann test. Plain X-ray films of the skull revealed much thickening and sclerosis of the bone in the right fronto-parietal region. Tangential views revealed superficial erosion of external lamina. Here and there in the sclerotic bone small areas of rarefaction were seen. The clinical diagnosis was sarcoma of the skull.

Treatment consisted of wide incision of the scalp and skull in circular fashion, the incised area being four inches in diameter. It was then found that the *dura mater* beneath the growth had been invaded over an area of five by five centimetres. The affected dura was removed with a wide area of healthy dura. No attempt had been made as yet to fill in the bony defect, and it might not be necessary or even advisable in view of the patient's age and secluded existence to submit her to further surgical treatment. The dural gap was closed with a *fascia lata* graft taken from the thigh, and the huge hiatus left after removal of the tumour, skull and scalp was covered by securing a pedunculated scalp flap (a circle 10 centimetres in diameter) from the left and posterior region of the scalp. The donor site of the scalp was later covered with pinch grafts taken from the skin of the thigh.

The tumour was found on microscopic examination to be a sarcoma. It had infiltrated the skin and the underlying *dura mater*. Convalescence had in every way been satisfactory. The wounds had healed well and there was no sign of local recurrence of the growth.

Left-Sided Subdural Hæmatoma (Traumatic) with Homolateral Hemiplegia.

Dr. Morgan's second patient, a man, aged sixty-two years, had been rendered unconscious when knocked off his bicycle in a street accident three weeks previously. He was observed in the casualty department for some hours and allowed to go home. There were scalp injuries in the left frontal and parietal areas. He was readmitted to hospital three days later because he had become drowsy; left hemiparesis had developed and the left plantar reflex had become extensor. He was then found to be suffering from diabetes (he had a high renal threshold) and hypertension. Indeed he had had a mild cerebral thrombosis three years earlier causing temporary left hemiparesis. His condition improved for two weeks, but then he again became more drowsy and mentally confused. (However, the fasting blood sugar level remained steady.) The left hemiparesis involved the face and hand chiefly, the left plantar reflex became more definitely extensor, and examination of the optic fundi revealed hæmorrhages and exudates. The blood pressure rose from 170 to 200 millimetres of mercury, systolic, but the pulse

rate did not become slower. Plain X-ray films of the skull revealed no calcification of the pineal gland.

A diagnosis was made of subdural hæmatoma, and indeed when burr holes were made a fluid subdural hæmorrhage was found to extend over the whole of the left hemisphere.

Dr. Morgan said that the occurrence of the hemiparesis on the same side as the hæmorrhage could be explained by compression of the right half of the brain stem against the right edge of the *tentorium cerebelli*, towards which it was displaced by the incarceration of the uncus of the left temporal lobe between the brain stem and the tentorial edge of the left side.

Carotid Aneurysm within the Cavernous Sinus Presenting with Profuse Epistaxis.

Dr. Morgan next showed a female patient, aged forty-eight years, who had been referred from Gippsland by Dr. G. Baldwin because of alarmingly profuse epistaxis. It had commenced a year before, but had become more frequent during the month before her admission to hospital. The bleeding always occurred from the left nostril and always began and ended suddenly. Volumes up to 28 ounces were reported to have been lost in as short a space as five minutes, and she had required several transfusions of blood. She also complained of flushing of the left side of the face and latterly of a dull ache in the left side of the head.

On examination, the patient was found to have various skeletal deformities including defective ossification of the upper third of the left humeral shaft, and a rudimentary left thumb, both well demonstrated by X-ray studies. Apart from a pterygium over the right eye, which had been present as long as she remembered, there were no abnormal findings in the nervous system, or in the naso-pharynx. The cerebro-spinal fluid was normal in all respects and the blood failed to react to the Wassermann test. Plain X-ray pictures of the skull revealed considerable erosion around the pituitary fossa and sphenoidal sinus. Left common carotid arteriography revealed a large aneurysm of the carotid artery within the left cavernous sinus. Obviously it had ruptured periodically into the sphenoidal air sinus. Dr. Morgan said it was strange that none of the nerves in the cavernous sinus showed signs of involvement.

On December 10, 1951, the left internal carotid artery had been ligated and divided in the neck. No further hæmorrhages had occurred, and no new signs had developed since that time.

Intracranial Aneurysm Simulating Tumour.

Dr. Morgan's next patient was a labourer, aged fifty-five years, who complained of having suffered three epileptic fits during the past six months. His wife had observed slight weakness of the right side of the face and some irregularity in his handwriting during the previous five months. She also vouchsafed the information that when walking he had tended to veer a little to the left for three months past. He had lost seven pounds in weight in five months.

Physical examination of the patient revealed the following. His memory for recent events and his ability to perform simple calculations were impaired. There was right facial paresis of the upper motor neuron type, and slight motor weakness and increased tone were noted in the right upper limb. The right supinator jerk was increased. The cerebro-spinal fluid was slightly xanthochromic and under normal pressure. It contained 70 milligrammes of protein *per centum*. The Wassermann test failed to produce a reaction. Plain X-ray films of the skull revealed widened meningeal vascular channels on the left side, and encephalographic examination revealed displacement of the ventricular system to the right. There was a large filling defect of the inferior and forward part of the left lateral ventricle and the adjacent part of the third ventricle.

At this stage the diagnosis was inferior left frontal tumour, probably mengingioma. However, arteriography was performed on the left common carotid artery and disclosed a huge aneurysm of the intracranial internal carotid artery springing from it just above the clinoid process on the left side. The diameters of the aneurysmal sac were 18 millimetres (vertical), 15 millimetres (sagittal) and 14 millimetres (transverse). The irregular filling of the sac indicated extensive spontaneous thrombus formation.

Treatment consisted of ligation and division of the left common carotid artery, and later of the left internal carotid artery on May 8, 1952, followed in each case by a short course of heparin and dicoumarol therapy. Dr. Morgan said that since operation the right hemiparesis had disappeared, memory and power to calculate had improved and the patient had returned to work.

Intramedullary Simple Cyst of the Spinal Cord.

The next patient shown by Dr. Morgan was a bookmaker, aged forty-nine years, who had been in good health until four months before his admission to hospital on February 9, 1952, when he slipped and fell on the buttocks. Thereafter he complained of intermittent, spontaneous pain in the front of the thighs and the inner aspect of the knees. For three months weakness, involuntary twitching and numbness of the lower limbs were noted.

On examination of the patient the following abnormalities were found: (*a*) severe spastic paraplegia, with increased tendon reflexes, ankle clonus and extensor plantar reflexes; (*b*) below the tenth thoracic spinal segment, loss of vibration sense; (*c*) loss of sensation for pain and temperature in the lumbar and upper three sacral segments on the right side and in the upper three lumbar segments on the left side; (*d*) impaired sense of position in the lower limbs and the presence of Romberg's sign.

X-ray films of the lumbar and thoracic parts of the spine revealed no abnormality. Gastric analysis and examination of a blood film gave normal findings, and the blood failed to react to the Wassermann test. The Queckenstedt test indicated a partial subarachnoid block. The cerebro-spinal fluid contained 100 milligrammes of protein per 100 millilitres. Myelography demonstrated blockage of the lipiodol column at the level of the tenth thoracic vertebra.

A diagnosis of intramedullary tumour of the spinal cord was made.

Laminectomy with exposure of the spinal cord was performed on February 28 at the level of the tenth thoracic vertebra. No extramedullary lesion could be found. The spinal cord was larger than normal and bulging backward. A hypodermic needle was inserted into the cord and 1·5 millilitres of slightly turbid fluid were aspirated. The fluid contained much protein and some debris difficult to identify accurately. No specific findings were recorded. After the operation the patient was able to walk well and easily, whereas before he could take only a few steps with great difficulty. The twitchings and the pain had ceased. Sensory dysfunction had decreased slightly and was showing a slowly progressive change for the better.

Large Suprasellar Aneurysm of the Left Internal Carotid Artery with Complications.

Dr. Morgan then showed a male patient, aged twenty years, who dated the onset of his symptoms from a motor bicycle accident, which had occurred two years before his admission to hospital; he had suffered facial injuries and a probable fracture of the skull. Since then he had had intermittent frontal headache, and in the past five months blurring of vision, near vision being particularly affected and vision at night being worse than vision during the day. The right eye was more severely affected than the left. During the past three months he had suffered occasional dizzy turns. He needed to shave only once a week.

On examination of the patient, his skin was soft and pale and relatively hairless. The heart, chest and blood were normal. He had bilateral optic atrophy; visual acuity (Snellen) was 6/15 on the left and 6/30 on the right. Investigation of the visual fields revealed left homonymous hemianopia. There were no other signs. The blood failed to react to the Wassermann test. A plain X-ray film of the skull revealed a calcified arc, concavity upward, of radius 1·5 cubic millimetres in the suprasellar region. The clinical diagnosis rested between a suprasellar cyst and an aneurysm of the internal carotid artery, probably on the right side, since the right eye was chiefly affected and the homonymous hemianopia to the left. The arteriogram of the right carotid artery failed to reveal an aneurysm, but revealed lateral and upward displacement of the carotid artery.

Exploration, by right frontal craniotomy, demonstrated a large aneurysm the size of a golf ball occupying the suprasellar region. A left carotid arteriogram was taken and the huge aneurysm shown to originate from the left internal carotid artery. The left common carotid artery was first ligated and divided. Three months later the left internal carotid was ligated and divided in the neck. Both operations were well borne. Dr. Morgan said that the headache had been relieved, and the visual failure, which progressed after the common carotid ligation, had halted since the internal carotid ligation.

Dumb-Bell Neurofibroma.

Dr. Morgan finally showed a school-boy, aged sixteen years, who gave a history of an operation for removal of a tumour in the left supraclavicular region five years earlier. The tumour was tender and pressure on it caused pain to

radiate into the left upper limb. After the operation the left shoulder was weak, especially for abduction movements. The weakness lessened but did not completely recover under treatment by immobilization of the shoulder and physiotherapy. In February, 1952, he complained of a recurrence of the swelling and of transient episodes of weakness of the left lower limb over a period of five months.

On examination of the patient, the following abnormalities were found. There was a firm ovoid swelling the size of a pullet egg in the left supraclavicular triangle lying deep to skin and deep fascia, beneath the incision of the operation referred to, and in the line of the upper nerves of the brachial plexus, immovable in either direction, but freely movable in a line at right angles to them. Upper brachial plexus palsy was present. There was weakness of the lower limbs, with increased tendon reflexes and extensor plantar reflexes. The Queckenstedt test indicated complete obstruction to the flow of the cerebro-spinal fluid. The cerebro-spinal fluid contained 120 milligrammes of protein per 100 millilitres. X-ray examination of the cervical part of the spine revealed enormous expansion by pressure atrophy of the intervertebral foramina between the third, fourth and fifth cervical vertebræ. A myelographic examination was carried out. Lipiodol was introduced into the *cisterna magna*. There was a complete hold-up of the lipiodol at the level of the middle of the third cervical vertebra. The radiographic appearance of the lipiodol column was the "gendarme's cap", which was typical of an extramedullary tumour. A diagnosis of dumb-bell neurofibroma was made.

On March 7, 1952, cervical hemilaminectomy was carried out at the level of the third, fourth and fifth cervical vertebræ; the tumour, circumscribed and now invasive, was found. It was clearly differentiated into extradural, intradural and intervertebral parts, all of which were removed, together with the *dura mater*, through which the tumour passed, and an adjacent healthy fringe. The intradural part was bulbous in shape, the extradural part was discoid, and from this base the conical intervertebral portion pushed its way through the greatly expanded intervertebral foramina. The wound was closed securely and with great care to prevent a cerebro-spinal fistula, since the dura was deficient at the site of extirpation of the tumour.

On March 20 the cervical extension of the tumour was removed by opening the original supraclavicular incision and dissecting the growth from the intervertebral foramina, along the upper roots of the brachial plexus to a distance of six centimetres from the foramina. In this dissection it was impossible to differentiate tumour from nerve roots. At the conclusion the third and fourth intervertebral foramina were bare of nerve tissue, and cerebro-spinal fluid leaked from them. The result was complete restoration of power in the lower limb. There was a complete lesion of the fourth and fifth cervical roots.

Out of the Past.

In this column will be published from time to time extracts, taken from medical journals, newspapers, official and historical records, diaries and so on, dealing with events connected with the early medical history of Australia.

ON THE TREATMENT OF DIPHTHERIA BY THE LOCAL APPLICATION OF PERMANGANATE OF POTASH, BY JOHN DAY, M.D.[1]

[*Australian Medical Journal*, February, 1864.]

The prevalence of diphtheria in Geelong during the past three months has afforded me a good opportunity of testing the efficacy of Permanganate of Potash in that disease. I am indebted to a paper by Dr. Charles Bell of Edinburgh for the suggestion (Vide Medical Review March 1861). Since the 18th of November I have treated fifteen cases in this way, trusting almost entirely to the local application of a weak solution of Permanganate of Potash. Thirteen are perfectly restored to health and the two remaining cases

[1] From the original in the Mitchell Library, Sydney.

DISEASES NOTIFIED IN EACH STATE AND TERRITORY OF AUSTRALIA FOR THE WEEK ENDED JANUARY 24, 1953.[1]

Disease.	New South Wales.	Victoria.	Queensland.	South Australia.	Western Australia.	Tasmania.	Northern Territory.[2]	Australian Capital Territory.	Australia.[3]
Acute Rheumatism	..	3	..	..	..	..	..	..	3
Amœbiasis	..	..	..	..	..	..	..	..	..
Ancylostomiasis	..	..	..	..	1	..	..	..	1
Anthrax	..	..	..	..	..	..	..	..	..
Bilharziasis	..	..	..	..	..	..	..	..	..
Brucellosis	..	..	..	..	..	..	..	..	..
Cholera	..	..	..	..	..	..	..	..	..
Chorea (St. Vitus)	..	..	..	..	..	..	..	..	..
Dengue	..	..	..	..	..	..	..	..	..
Diarrhœa (Infantile)	5(5)	..	5(1)	..	..	..	..	..	10
Diphtheria	11(8)	2(1)	11(4)	..	1	1	..	..	26
Dysentery (Bacillary)	..	1(1)	..	..	1(1)	..	..	..	2
Encephalitis	..	3(1)	..	3(3)	..	..	..	..	6
Filariasis	..	..	..	..	..	..	..	..	..
Homologous Serum Jaundice	..	..	..	..	..	..	..	..	..
Hydatid	..	..	..	..	..	..	..	..	..
Infective Hepatitis	..	7(2)	..	..	9(8)	..	..	..	16
Lead Poisoning	..	..	..	..	..	..	..	..	..
Leprosy	..	..	..	..	..	..	..	..	..
Leptospirosis	..	..	1	..	..	..	..	..	1
Malaria	..	..	1	..	..	..	..	..	1
Meningococcal Infection	2(2)	2(2)	..	..	..	1	..	..	5
Ophthalmia	..	..	..	..	..	..	..	..	..
Ornithosis	..	..	..	..	..	..	..	..	..
Paratyphoid	..	..	..	..	..	..	..	..	..
Plague	..	..	..	..	..	..	..	..	..
Poliomyelitis	30(12)	9(6)	7(1)	16(13)	1	12(2)	..	..	75
Puerperal Fever	..	..	3	..	..	..	..	..	3
Rubella	..	37(23)	..	..	2(1)	..	..	..	39
Salmonella Infection	..	..	..	..	..	..	..	..	..
Scarlet Fever	7(2)	15(7)	4(1)	3(3)	..	2	..	..	31
Smallpox	..	..	..	..	..	..	..	..	..
Tetanus	..	1	..	..	..	..	..	..	1
Trachoma	..	..	..	..	..	..	..	..	..
Trichinosis	..	..	..	..	..	..	..	..	..
Tuberculosis	34(22)	38(31)	23(11)	11(5)	3(3)	7(4)	..	1	117
Typhoid Fever	..	1(1)	1	2(2)	..	..	..	..	4
Typhus (Flea-, Mite- and Tick-borne)	..	..	1(1)	..	..	..	..	..	1
Typhus (Louse-borne)	..	..	..	..	..	..	..	..	..
Yellow Fever	..	..	..	..	..	..	..	..	..

[1] Figures in parentheses are those for the metropolitan area.
[2] Figures not available.
[3] Figures incomplete owing to absence of returns from the Northern Territory.

are convalescent. With the exception of an emetic and an aperient if necessary I give little or no medicine, nor do I use any caustic application to the throat.

The immediate relief in some of these cases and the steady and rapid improvement in all, warrant me in recommending it to my professional brethren as a most valuable remedy.

My friend Dr. Carstairs has tried this plan of treatment in six cases with the most satisfactory results. He however gave chlorate of potash and tincture of sesquichloride of iron internally.

Permanganate of Potash is prepared in a pure form for the use of the profession by Mr. Condy the well known discoverer of Condys Disinfecting Fluid which is a solution of Permanganate of Potash. Dr. Bell recommends the use of Condy's Disinfecting Fluid in proportion of one part to eight of water which is to be applied to the throat by means of a camel hair pencil several times a day.

Not knowing the exact strength of Condys Fluid I generally prescribe from a quarter of a grain to a grain in half a pint of pure water to be used either with a brush or as a gargle.

Geelong,
January, 1864.

Post-Graduate Work.

SEMINARS AT ROYAL PRINCE ALFRED HOSPITAL.

THE following seminars will be held at the Royal Prince Alfred Hospital, Camperdown, New South Wales, during the first half of 1953. Each seminar is held in the Scot Skirving (A2) Lecture Theatre on Friday between 1.15 and 2.15 p.m. and is followed by a clinico-pathological conference from 2.30 to 3.30 p.m., unit staff rounds from 3.30 to 4 p.m. and a demonstration of patients from 4.30 to 5.30 p.m. All medical graduates are invited to attend.

March 6, hæmatology section, "Blood Picture in Glandular Fever". March 13, diabetic clinic, "Diabetic Neuropathy", Dr. R. D. Lawrence, London. March 20, no seminar. March 27, gastro-enterology section, "Post-Gastrectomy States".

April 3, no seminar. April 10, public health section, lecture by Sir Allen Daley (London). April 17 and 24 and May 1, neurology section, "A Restatement on Encephalitis", "Cerebral Disorders of Vision" and "Disorders of Language", Dr. Macdonald Critchley (London).

May 8, radiology section, "The Association and Correlation of Congenital Bone Dystrophies", Dr. H. R. Sear. May 15, pædiatrics section, "Pink Disease". May 22, pathology section, clinico-pathological conference. May 29, cardiology section, "Mitral Stenosis".

June 5, hæmatology section, "Significance of the Platelet Count and the Erythrocyte Sedimentation Rate". June 12, gastro-enterology section, "The Usefulness of the Aspiration Liver Biopsy Needle". June 19, thoracic section, "The Estimation of Pulmonary Function in Thoracic Disease". June 26, pædiatrics section, "Disturbances of Growth in Childhood".

Deaths.

THE following deaths have been announced:

SCOTT.—George Ernest Mueller Scott, on January 29, 1953, at South Yarra, Victoria.

SPEEDING.—Keith Rennick Speeding, on February 4, 1953, at Moorabbin, Victoria.

MCCARDEL.—Edward Leo Anthony McCardel, on February 8, 1953, at Benalla, Victoria.

HOLDER.—Sydney Ernest Holder, on February 9, 1953, at Adelaide.

TEMPLEMAN.—Colin Gordon Templeman, on February 13, 1953.

CUMMINS.—George Cummins, on February 14, 1953, at Melbourne, Victoria.

Notice.

LAENNEC SOCIETY.

A CLINICO-PATHOLOGICAL MEETING of the Laennec Society will be held at the Repatriation General Hospital, Concord, on Wednesday, March 25, 1953, at 7.45 p.m. All physicians and surgeons interested in thoracic disease are cordially invited to attend.

Diary for the Month.

MARCH 3.—New South Wales Branch, B.M.A.: Organization and Science Committee.
MARCH 4.—Victorian Branch, B.M.A.: Clinical Meeting.
MARCH 4.—Western Australian Branch, B.M.A.: Council Meeting.
MARCH 5.—South Australian Branch, B.M.A.: Council Meeting.
MARCH 6.—Queensland Branch, B.M.A.: General Meeting.
MARCH 10.—New South Wales Branch, B.M.A.: Executive and Finance Committee.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment mentioned below without having first communicated with the Honorary Secretary of the Branch concerned, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

New South Wales Branch (Medical Secretary, 135 Macquarie Street, Sydney): All contract practice appointments in New South Wales.

Victorian Branch (Honorary Secretary, Medical Society Hall, East Melbourne): Associated Medical Services Limited; all Institutes or Medical Dispensaries; Australian Prudential Association, Proprietary, Limited; Federal Mutual Medical Benefit Society; Mutual National Provident Club; National Provident Association; Hospital or other appointments outside Victoria.

Queensland Branch (Honorary Secretary, B.M.A. House, 225 Wickham Terrace, Brisbane, B17): Brisbane Associated Friendly Societies' Medical Institute; Bundaberg Medical Institute. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL or position outside Australia are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.

South Australian Branch (Honorary Secretary, 178 North Terrace, Adelaide): All Contract Practice appointments in South Australia.

Western Australian Branch (Honorary Secretary, 205 Saint George's Terrace, Perth): Norseman Hospital; all Contract Practice appointments in Western Australia. All government appointments with the exception of those of the Department of Public Health.

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